PROCEDURE IN EXAMINATION OF THE LUNGS

ARTHUR F. KRAETZER

OXFORD MEDICAL PUBLICATIONS

PROCEDURE IN EXAMINATION OF THE LUNGS

OXFORD MEDICAL PUBLICATIONS

Procedure in Examination of the Lungs

With Especial Reference to the Diagnosis of Tuberculosis

By

ARTHUR F. KRAETZER, M.D.

CHIEF, MEDICAL DEPARTMENT, NEW YORK SKIN AND CANCER HOSPITAL; INSTRUCTOR IN MEDICINE, CORNELL UNIVERSITY; FORMERLY, CHIEF TUBERCULOSIS OUT-PATIENT DEPARTMENT, BELLEVUE HOSPITAL, AND ASSISTANT ADJUNCT ATTENDING, TUBERCULOSIS SERVICE, BELLEVUE HOSPITAL.

WITH A FOREWORD BY

JAMES ALEXANDER MILLER, M.D.

NEW YORK

OXFORD UNIVERSITY PRESS

COPYRIGHT, 1930
BY OXFORD UNIVERSITY PRESS
NEW YORK, INC.

PRINTED IN THE UNITED STATES OF AMERICA

Dedicated to

MY WIFE

FOREWORD

The diagnosis of pulmonary tuberculosis is one of the real problems in the teaching of medicine, not only in the medical school but also for the practitioners of medicine who have already graduated. Tuberculosis is one of the most common diseases, and yet it is the very frequent experience that many cases are neglected and undiagnosed long after it should have been possible by the means of diagnosis at our command.

Dr. Kraetzer has struck at one of the most important contributing causes to this unfortunate situation, that is, the method of approach to the problem which is given to the medical student during his under-graduate course. His little book does not claim to contain very much that is new, but the method of treatment is quite original and, based upon long experience with students, it is essentially practical rather than theoretical. Tuberculosis, viewed from this standpoint at the very beginning of the student's approach to it, should be a very much simpler problem than it has turned out to be in the past.

There is really nothing very complicated in the method of making physical examinations of the chest or arriving at correct conclusions. Certainly in nine-tenths of the cases this should be possible in the hands of the general practitioner, and only in the exceptional case should it be

needed to bring more expert advice and more extensive experience to bear on the problems involved. Many of our text-books have made the examination of the chest far too complicated, and Dr. Kraetzer has made a real contribution in simplifying the methods involved.

It must be borne in mind, however, that physical signs are by no means always the most important evidence in making a diagnosis of pulmonary tuberculosis. In many, if not the majority of cases, the diagnosis should be suspected at least, if not really made, from the history alone, and in so many cases does it occur that physical signs are very scanty or absent or perhaps not pathognomonic, that if a student or physician acquires a habit of relying upon physical signs for diagnosis, many mistakes will result. As Dr. Kraetzer well points out, it is the question of suspicion aroused by the points brought out in the story which should lead eventually to the correct diagnosis, and this suspicion must not be lulled to sleep in the absence of unequivocal physical signs. Repeated examination, careful study of the story, x-ray studies and sometimes prolonged observation may be needed.

It is a very real problem which faces the medical profession and for the solution of which it alone is responsible. Too often the criticism of the laity against the profession for failure to make early or correct diagnoses is well founded. Dr. Kraetzer's little book is an important step in the right direction and should prove very useful to students.

James Alexander Miller, M.D.

CONTENTS

CHAPTER		PAGE
	FOREWORD	vii
	INTRODUCTION	xi
I.	THE ACTUAL SOUNDS	1
II.	THE EXAMINATION OF THE CHEST	11
III.	INSPECTION	12
IV.	PALPATION	16
V.	THE HEART IN RELATION TO PULMONARY EXAMINATION	20
VI.	PERCUSSION	23
VII.	AUSCULTATION	39
VIII.	DIMINISHED INTENSITY OF BREATH-SOUNDS	51
IX.	BRONCHO-VESICULAR AND BRONCHIAL BREATH-SOUNDS	84
X.	RALES	86
XI.	THE SIGNS OF TUBERCULOSIS	108

INTRODUCTION

The purpose of this book is to furnish the student with a correct introduction to the technique of physical examination and diagnosis of the lungs. The conventional teaching of this subject, in text-book and class, brings the student at the very start face to face with two serious handicaps.

In the first place, the conventional teaching method is the exact opposite of the actual process of examining a chest. Conventional instruction is deductive. Actual examination is inductive. Conventional instruction starts with general principles and works down. The student is crammed with a confusing mass of facts and near-facts and then is expected to single out in a given case the particular facts he needs and apply them. The actual examination starts perforce with the gathering of particular findings and then works upward to an inductive conclusion.

Conventional texts take up two topics; (1) the description of manual and aural technique in the discovery of physical signs, (2) the description of the diseases in terms of the physical signs. The assumption then is that the work is done, and that all the student has to do is to read up on these topics and apply the information contained therein. If he cannot do it, so much the worse for him.

However, with the elucidation of these two topics the

work is by no means done. Before the student can diagnose a chest, he must effect an essential synthesis of technique on the one hand and descriptive knowledge of disease on the other. This synthesis is not taken up in the average text-book. It is an inductive process, something more vital than mere facts, a kinetic mental procedure that must be learned, before diagnosis can be carried out.

This book attempts to develop in teaching the natural process that is followed in practice. It is curious that, three hundred years after the time of Francis Bacon, the old academic deductive method still should be used in practically all departments of teaching, in preference to the natural inductive method, which is the only real means of acquiring knowledge of any kind. The technique of instruction must approximate the technique of acquiring facts in general, and that technique is necessarily inductive.

In the second place, the student is thrown very early into a confused and deprecatory state of mind. Many things conspire to bring about this unhealthy condition. First is the inherent clumsiness of the deductive method itself, which is all that the conventional presentation gives. Furthermore, the student often is discouraged by the impatience and intolerance of his instructors, who find fault with what they are pleased to call his stupidity. The knowledge of the instructors has been hard-won, and human vanity, to which instructors are not immune, sometimes tempts them to convey the impression that chest-

diagnosis is reserved for the few elect. This does not contribute to the student's self-confidence, and as a result of all these psychological hazards he becomes considerably mixed. I shall never forget a fellow-student "listening" to a heart with the stethoscope not in his ears, but hanging around his neck, and swearing that he heard the murmur. At the time, this was considered extremely humorous, but to myself it was a very tragic example of utter loss of morale. That student is today an excellent gynecologist, but he has forsworn the stethoscope as something mysterious and possibly not quite holy.

Nothing in Medicine is worse done than the early diagnosis of tuberculosis, and one of the factors that contributes to this is, I am sure, the unnaturalness and obscurity of early training. Not only the method, but also the matter, is vague. Nothing could be more confused, for example, than the classical and utterly obsolete classification of rales. The terms crepitant and subcrepitant are entirely ambiguous. They have no place in modern clinical terminology.

In this book I have attempted to lay down an inductive method of teaching that parallels the inductive method of examining each and every case. In no sense does it pretend to be a diagnostic hand-book. Its purpose is the development of method and nothing else. As regards presentation, I have reproduced as accurately as I could, the actual conversations with students in the clinic, and have forsworn at the start everything that pertains to conventionality of treatment.

The purpose of examination of the lungs is obviously the diagnosis of lung conditions, and, in a book dealing with this subject, every pulmonary problem must find a place. In the delineation of diagnostic method a book must be an organic unit, and from the purely teaching point of view one disease is as important as another. From the practical aspect, however, the crowning glory of the work in hand would be to facilitate the early diagnosis of tuberculosis, and upon this element especial emphasis is laid.

Procedure in Examination of the Lungs

Chapter I

THE ACTUAL SOUNDS

Areas to be Compared.
- *Percussion.*
  - *a.* Resonance or duration of sound.
  - *b.* Pitch of percussion note.
- *Auscultation.*
  - *a.* Intensity or relative loudness.
  - *b.* Length of expiratory sound.
  - *c.* Pitch of inspiratory sound.
  - *d.* Quality of breath-sounds.

Origin of the Sounds.

Cavernous and Amphoric Breathing.

Technic of Percussion.

The purpose of this chapter is to furnish the medical student with an actual, objective critique of his percussive and auscultatory findings, and to give not an arbitrary but a comparative and synthetic description of what he hears. It is in no sense a syllabus of physical diagnosis but is merely a practical method of developing technique and giving the student a system of defining a given sound, that may confuse him, by comparing it with other known areas on the chest. The regular routine of inspection, palpation, etc., and their diagnostic significance come

later. This chapter deals merely with the elucidation of sounds.

First of all, pick out some member of the family to practise on at home and follow this routine rigidly. Take it easy, one point at a time, as laid down in the outline, and do not listen too hard. Straining the attention often vitiates results. Remember that these things are really very easy to hear, at least in the vast majority of cases.

Compare the following four points on the normal individual (see Fig. 1), in the following order:

1. A point rather low in the right axilla; do not use the left, because gastric tympany may be present, and the heart-sounds may cause confusion,
2. The left supra-clavicular region,
3. The right supra-clavicular region,
4. The side of the neck.

Observe and compare each point with respect to I. Percussion, II. Auscultation.

I. Percussion

Take up the following points in the order given:

(a) *Resonance, or duration of sound.* — The resonant sound actually lasts longer than the dull sound. If in doubt, simply ask the question, "Which sound lasts longer?" A very little practice will show this to be an absurdly easy test.

(b) *Pitch of the percussion note.* — This is perhaps a little more difficult. Some ears are attuned more deli-

cately to slight changes in pitch than others, but the difference is not sufficient for the student to develop an inferiority complex over it and decide that he will never be able to diagnose tuberculosis. A simple rule to remember is that the duller of two notes is always the higher pitched, and, knowing this, you can always check one against the other. If, under " Ia ", you have decided that a given note is duller than another, then the former is higher pitched. Try to sing the two notes and keep at it until you are convinced of the difference. The axilla will give a resonant, or " long ", and low-pitched note. The left supra-clavicular region will have resonance, but less of it. Its note will not last quite so long, and it will be slightly higher pitched. Normally, the difference is not of sufficient degree to call the left apex actually dull. Over the right apex, the difference will be even more marked, the note duller, or shorter, and still higher pitched. It still maintains, however, a slight amount of resonance or " ring ", lasting beyond the moment of actual percussion. Percussing the side of the neck shows flatness, or the superlative of dullness. It gives no ring lasting over and beyond the instant of the stroke, and it is still higher pitched.

In other words, the dullness increases, and the pitch rises, in the order 1, 2, 3, 4. Keep at it until you are sure of these differences. You will be surprised how easy it is. Once having gained this ability, realize that there are no subtler shadings in the whole field of percussion.

II. Auscultation

Compare the points, 1, 2, 3, 4, in that order, with respect to:

(a) *Intensity, or relative loudness.* — As a rule, intensity of breath-sounds will increase in the order 1, 2, 3, 4. "Four" will always be louder than all the rest, and "three" will always be louder than "two". "One", however, sometimes may be louder than two or three. Remember that anybody can decide which of two sounds is the louder. You do not have to be a Laennec for that.

(b) *Length of expiratory sound.* — This, without exception, will increase in the order 1, 2, 3, 4. Over "one" you may hear no expiratory sound whatever, and this is perfectly correct. At times, particularly in young individuals, you may hear a very considerable expiratory sound. This is not to be confused with bronchial breathing. The distinction soon will be made clear.

(c) *Pitch of inspiratory sound.* — Listen to the inspiratory sound and immediately remove the stethoscope so as not to hear the expiration, shifting it to the area next in order in time to catch the succeeding inspiration. The pitch will rise in the order 1, 2, 3, 4. At this point one precaution is necessary. As was mentioned above, "one" may at times be more intense or louder than "two" and "three". Do not mistake increased intensity for relatively higher pitch. It is an easy pit to fall into. We hear loud sounds more readily than less loud sounds, and high-pitched sounds more readily than low-pitched. Be-

cause of this factor of easier audibility, common to loud sounds and high-pitched sounds, we may jump to the false conclusion that a relatively louder (more intense) sound is relatively higher pitched, whereas it is merely louder and actually may be lower pitched. The difference in pitch of the inspiratory sounds, taken by itself, is of little value diagnostically. As an exercise in technique, however, its recognition is of the greatest importance.

The foregoing three properties of breath-sounds are quantitative in their nature and are defined according to quantitative standards. The sound is louder or less loud. It is longer or shorter. It is of higher or lower vibration-frequency, i.e., higher or lower pitched. The next property, as its title indicates, is not quantitative but qualitative. The variations of this property are " sui generis " and simply must be learned as such. Even here, however, there is a quasi-quantitative transition of one quality into another.

(d) *Quality of breath-sounds,* or degree of approximation to the hollow quality of " four ", which is the tracheal sound. — " One " will possess none of this quality. The quality of " one " is known as vesicular, because it is supposed to arise in, or be transmitted through, unimpaired air-vesicles. It may be described best as a gentle, unobtrusive rustle, as of myriads of microscopic leaves. If you attempted to represent it graphically, you would do so with a multitude of minute dots, or stippling, if sound ever can be visualized as being stippled. The tracheal tone, however, over " four ", is a purely homogeneous, hol-

low, "unstippled" sound. These, of course, are figures of speech and very poor figures of speech. The differences in quality are impossible to describe in words, but are very easy to detect with the stethoscope. Now, "one" possesses none of the quality of "four". "Two" possesses a very little of it. "Three" possesses a very perceptible lot of it. Again we see a very definite progression from one to four.

Now for the synthesis of the above. "One" shows vesicular breathing. It is characterized by a generally comparatively low intensity or loudness, a generally inconsiderable or absent expiratory sound, a low-pitched inspiratory sound with a gentle, rustling, "vesicular" quality. "Four" shows tracheal, or for our present purposes, exaggerated bronchial breathing. Its essential characteristics are a markedly prolonged expiratory sound and a distinctive hollow quality. These two ear-marks are easy to make out. A third essential characteristic, less easy to distinguish at first, is heightened pitch. An accidental and very inconstant characteristic of bronchial breath-sounds is increased intensity or loudness. It must never be forgotten that frequently this does not obtain. The typical bronchial breathing of pneumonia may be very loud, but on the contrary it may be so remarkably suppressed as to be almost inaudible. In this event, the other lung may show a compensatory, exaggerated, vesicular breath-sound, with increased intensity and considerable prolongation of expiratory sound, and this may be mistaken for bronchial breathing, unless its other

characteristics, low pitch and absence of hollow quality, are noted. Simply call up to your mind the little critique laid down above and separately compare each element of the breath-sounds. Suppressed, almost inaudible bronchial breathing can be recognized easily by a simple trick, the use of the whispered voice, which, you will remember, is nothing more or less than a reinforced expiratory sound. Some patients whisper during inspiration. This must be guarded against.

We have now described vesicular breathing, heard at "one", and practically bronchial breathing, heard at "four". Broncho-vesicular breathing is simply a transition between one and four and is heard normally at "three", the right apex. The sound at "two", the left apex, is in turn transitional between one and three and is indeterminate, not quite vesicular, not quite broncho-vesicular.

If you learn to make the above distinctions, and fifteen minutes practice a night for two weeks will teach you, you will be capable of distinguishing the finest differences that occur in auscultation. To utilize this ability diagnostically, however, means much more training and practice, but the essential groundwork will be there. Disabuse your mind at the start from the notion that preternatural acuteness of hearing is needed. Establish the above discipline, and you will be in no danger of thinking that you know too much, and you will be saved from the even more tragic obsession that you are incapable of learning percussion and auscultation at all.

Origin of the Sounds

A word of warning to the beginning student well may be interpolated here. Do not, at this stage of the game, go in for learned and abstruse speculations on the physics or acoustics of the breath-sounds. It is very tempting to be side-tracked from the laborious acquirement of technic to the more imposing and much easier realms of speculation. Most of us prefer problems that cannot be solved, which take up in a pleasant way time that should be expended in toil and in which no honor can be lost, to problems that actually can be solved but take devilish hard work to do it. In the Middle Ages there was never any dearth of scholasts ready to use good time in debating about how many angels could dance at one time on the point of a needle, because it did not take any work, and nobody could prove you wrong anyway. There were only a few, however, who were willing to ruin their eyes grinding lenses or dissecting bugs, and they were not liked.

It is sufficient to say that in general there are two theories regarding the nature of the breath-sounds, and the proponents thereof regard each other like Guelphs and Ghibbelines. One idea is that the sounds actually arise in the lung tissue itself and vary with the condition of the latter. The other is that the sounds arise in the vocal cords and depend for their ultimate characteristics on the nature of the medium through which they are transmitted to the auscultator's ear. This latter is the generally accepted view, and there are certainly strong

points in its favor. The whispered voice, which is simply a reinforced expiratory sound, most assuredly arises in the vocal cords and not in the lungs. That it is inaudible over the normal pulmonary parenchyma and distinctly audible over an area of consolidation is an incontrovertible demonstration of its audibility or inaudibility being a definite function of the lung medium through which it is transmitted. The solid lung transmits it clearly to the ear. The spongy normal lung dampens it out of existence. Conversely, the laryngectomized or intubated or tracheotomized patient certainly has his full quota of circulating pulmonary air, yet no breath-sounds are to be heard over his lungs. His larynx has been removed or shunted off, and the source of the breath-sounds is gone. However, which theory is held to matters little for our present purpose.

Cavernous and Amphoric Breathing

Cavernous breathing is low-pitched, very hollow, rather "spooky"-sounding bronchial breathing. Amphoric breathing has the characteristics, essential and accidental, of bronchial breathing, that is to say it shows a prolonged expiratory element, it has a high pitch and a hollow quality. It may or may not be louder or less loud than the normal breath-sounds. In addition, and this gives it its definition, it has a distinctive musical quality, imitated by blowing over the mouth of a bottle. The whisper brings it out very distinctly.

Technic of Percussion

This chapter will end with a topic which is really more appropriate at the start, the description of a method for acquiring the technic of percussion. It was taught me by the late Dr. Charles Minor of Asheville, and if that master of chest diagnosis thought it worth while, the student need not find it without profit. The percussion stroke must be light, sharp, staccato, with an instantaneous return, absolute limpness of the wrist and absolutely no motion of the fore-arm, the pleximeter finger pressed firmly into the chest. Sit beside a table, with the pleximeter finger pressing on it. Balance a glass of water on the back of the right wrist and percuss. If you strike too hard, fail to make an instantaneous return, hold your wrist too stiff, or move your forearm, you dampen your note and are reminded of your error by the collapse of the glass of water. Five minutes of practice a night for a week, with a not unreasonable supply of glasses and water, will teach you perfect percussion.

Nothing in Medicine is more important to do right, or more badly done, than the early diagnosis of tuberculosis. It is quite possible that this inability on the part of so many physicians is based on a lack of confidence resulting from a superstition engendered in them as students, that pulmonary diagnosis is a superlatively difficult thing reserved for the few elect. The fore-going discipline, pedantic as it may seem in spots, is easy to acquire and will teach technic and confidence.

Chapter II

THE EXAMINATION OF THE CHEST

The examination of the chest is divided into four parts and is carried out in this order, inspection, palpation, percussion and auscultation. In the following account there will be described the various possibilities which may turn up during the examination, and an attempt will be made to duplicate the mental processes of the examining physician as he progressively accumulates his data. Whatever turns up under a given heading will be correlated with the possible findings which may have preceded it. The process thus becomes complicated by a sort of geometrical progression, but the result is a true piece of induction and is a fairly accurate approximation of what goes on, consciously and subconsciously, in the examiner's mind, although of course, in the latter case, more expeditiously. It is the sole way in which can be described that four-dimensional, bird's-eye view which correctly depicts method.

Chapter III

INSPECTION

General appearance.
Position of trachea.
Respiratory excursion.
Asymmetry of muscles.
Shape of chest.
Clubbing of fingers.

General Appearance

Have the patient sit in a comfortable, easy, erect position, directly facing you, with the hands in the lap and the muscles relaxed. Note the general nutrition and the type of physique. The classical "habitus phthisicus" is a lantern-jawed, long-necked, flat-chested piece of architecture. Very frequently, however, we find the exact reverse of this type, namely, a rounded, plump, well-nourished individual with a tendency toward feminine configuration and markings, what is sometimes referred to as the "hypo-pituitary type." When this type has tuberculosis, it seems to be of the active parenchymatous variety with rapid spread and cavitation and little tendency to arrest. It is extraordinary how well nutrition may be preserved in this type of patient, even with extreme involvement.

Next, note whether the patient "looks sick". A case

with considerable tuberculous consolidation sometimes may look surprisingly comfortable, whereas if the consolidation be that of pneumonia, he will always be very obviously ill, dyspnœic and febrile. If there are marked dyspnœa, cyanosis and collapse, with extraordinary and urgent distress, suspect pneumo-thorax or pulmonary embolism.

Position of Trachea

Next, note the position of the trachea. Drop an imaginary plumb-line from the notch in the thyroid cartilage and make a pen-mark where it strikes the episternal notch. It should strike almost precisely in the mid-line. Normally there may be an almost imperceptible deflection to the right, but if the deflection to the right is obvious, or if there is any deflection at all to the left, then the trachea is displaced, either from an ipso-lateral pull from scar tissue, or contra-lateral push from hydro- or pneumothorax. Lung tumors do not tend to displace the trachea. At times, however, the thyroid cartilage may be asymmetrical, and the plumb-line method may lead to wrong conclusions.

A more reliable and really easier method is to note the depressions between the lower end of the trachea and the lower ends of the two sterno-mastoids respectively. One of these depressions may look more filled in, the other correspondingly more scooped out. The filled-in side is the side toward which the trachea is displaced (Fig. 2).

Respiratory Excursion

Now note the respiratory excursion, particularly whether it is equal on both sides. Diminished excursion is easy to see, and in the absence of deformity always is indicative of disease on that side, such as:

1. Fibrositis of the intercostal muscles,
2. Pleurisy, dry or wet,
3. Tuberculosis, apical or extensive,
4. Pneumonia,
5. Pneumo-thorax,
6. Massive collapse,
7. Lung tumor,
8. Bronchiectasis,
9. Pulmonary abscess.

Asymmetry of Muscles

Look for asymmetry or atrophy of the muscles, particularly of the trapezius, upper intercostals, pectoralis major and rhomboideus major. According to Pottenger, pulmonary disease gives rise to a viscero-motor reflex expressing itself first in spasm of the muscles of the same segmental nerve-supply as that of the site of visceral involvement. This spasm is analogous to that over a diseased appendix. The initial spasm-producing viscero-motor reflex later gives place to a secondary atrophy-producing viscero-trophic reflex. Somatically, the expression of visceral disease is first, spasm; second, atrophy.

Shape of Chest

Note the costal angle, the degree of slant of the ribs and the ratio of the antero-posterior to the lateral diameter of the chest. An obtuse costal angle, an almost horizontal slant to the ribs and an antero-posterior diameter that is relatively great as compared to the lateral, are components of the so-called barrel-shaped chest characteristic of emphysema. An acute costal angle with sharply sloping ribs is characteristic of the so-called enteroptotic configuration and generally is accompanied by poor chest expansion.

Clubbing of Fingers

Now look for clubbing of the fingers. This almost always occurs in pulmonary abscess, generally occurs in bronchiectasis and less frequently occurs in tuberculosis. In the last-named condition it usually means extensive cavitation. It also is found in certain congenital cardiac diseases and at times may occur in such little understood conditions as so-called enterogenous cyanosis, where no pathology, either pulmonary or cardiac, can be demonstrated.

Chapter IV

PALPATION

Respiratory excursion.
Tactile fremitus.

Respiratory Excursion

Spread the fingers of both hands and place them lightly on both sides of the upper chest, the forefinger just below the clavicle, the other fingers approximately on the successive ribs. Have the patient breathe rather deeply in and out. In the young, normal, flexible chest, the second, third, fourth and fifth fingers will rise one after the other in the order given. In the senile or emphysematous chest, the fingers rise almost simultaneously. Note whether one side of the chest lags behind the other, and whether there is any inequality of excursion. Either of these findings is suggestive of disease on the side of delayed or limited excursion. Even a slight apical lesion may cause limited excursion on the diseased side.

Tactile Fremitus

In testing for tactile fremitus, apply to the chest that part of the palm which is just proximal to the fingers.

It seems to be more sensitive to vibration than the latter, possibly because it can be held against the chest more steadily and with less muscular strain. Use both hands, but use them one after the other. Do not apply them simultaneously. Shifting the same hand from one side of chest to the other takes too long. It makes you apt to forget just how strong the vibration felt. Have the patient say " ninety-nine " in a moderately loud, deep voice. The resulting vibration, felt by the hand on the chest, is known as tactile fremitus. Always compare symmetrical areas. Tactile fremitus is normal, increased, decreased, or absent. Normally it is slightly but definitely greater on the right than on the left side, particularly over the upper lobe. In this event it is incorrect to say that it is " increased " on the right, or " decreased " on the left. If the difference is more marked than you think it ought to be, then you have to make up your mind:

1. Whether the difference is more marked than normal. This you do by recalling normal chests previously examined,
2. Whether it is increased on the right, or
3. Whether it is decreased on the left.

This distinction may be hard to determine, but it is by no means purely academic. Of course, if the change is in the reverse order, i.e., if the tactile fremitus is greater on the left than on the right, the question is much easier to answer, and we know that we are dealing with an abnormal finding. In general, increases in tactile fremitus are more difficult to recognize as such and are more ambiguous in

significance than decreases. That is to say, a decrease is generally easier to determine and has more evidential significance than an increase.

Tactile fremitus may (or may not) be increased in:

1. Consolidation,
2. Cavity, generally, because of peripheral consolidation. Theoretically tactile fremitus should be decreased.

Tactile fremitus is decreased in:

1. Bronchial obstruction, asthma, foreign body, etc.,
2. Conditions insulating the lung from the chest-wall, such as pleural thickening or effusion, pneumothorax,
3. Aphonia,
4. Atalectasis, massive collapse.

If respiration is painful, place the palm of the hand over the site of the pain and have the patient breathe as deeply as he can. A palpable, scraping vibration means pleural friction. Its presence means pleurisy. Its absence does not rule it out. It may occur without any pain whatever, and it is most apt to be found in the axillary region. Dry pleurisy and fibrositis of the intercostal muscles may resemble symptomatically each other very closely. The presence of a friction-rub definitely establishes the diagnosis of the former condition. The differentiation of these two conditions, one an intrathoracic lesion of considerable menace, the other a banal myositis based on focal infection or grippe, is of the greatest importance. It is scandalous, however, how often

a mistake is made, the myositis generally being wrongly diagnosed as pleurisy. A factor contributory to this error is the fact that muscle-sounds are apt to be particularly in evidence over inflamed intercostal muscles and may be mistaken for a friction-rub.

Chapter V

THE HEART IN RELATION TO PULMONARY EXAMINATION

Mitral stenosis simulating pulmonary tuberculosis.
Displacement of heart away from diseased side.
Displacement of heart toward diseased side.

Before percussing the lungs, it is logical to interpolate the examination of the heart. In fact, this may just as well be done at the very start. In any event, it is essential to go over the heart *before any further examination of the lungs is made,* because by so doing, misconceptions and lost motions will be avoided. It is not pleasing to one's vanity, for example, in a case with cough, fever, hemoptysis, dyspnœa and night sweats to barge blindly into auscultating the lungs, and, on hearing a number of generalized moist rales, make a diagnosis of advanced tuberculosis with fatal prognosis, when examining the heart at the proper time would have disclosed the presystolic murmur of mitral stenosis and have established the lung condition at once as chronic passive congestion with entirely different outlook and treatment. One function of technic, secondary to the paramount one of serving the patient, is to spare ourselves as many intellectual upsets as possible. We get sufficient in the normal course of

events. Another and most essential reason for examining the heart before percussing and ausculating the lungs, is to determine the position of the cardiac borders and the apex-beat with possible pulmonary conditions in mind. If, without cardiac enlargement, the borders and the apex-beat are shifted, be quite sure that there is some pulmonary or pleural pathology. In such an event, refer back to your findings with respect to the position of the trachea and note whether this corresponds. The position of the trachea and the position of the heart are useful checks on each other. Rarely you may find the trachea displaced in one direction and the heart in the other, for example, a large cavity at the apex pulling the trachea over, and a large effusion at the base on the same side pushing the heart away.

Displacement of Heart away from Diseased Side

The heart is displaced away from the diseased side in:

1. Pleurisy with effusion,
2. Pneumo-thorax.

Displacement of Heart toward Diseased Side

The heart is displaced toward the diseased side in:

1. Marked fibrosis, and by inference, extensive cavitation, because it is generally only around large cavities that we get enough scar-tissue to exert a really considerable pull. This cavitation may be

the result of tuberculosis, bronchiectasis or abscess.

2. Massive pulmonary collapse. The key-finding in this post-operative condition is the displacement of the apex-beat toward the affected side. If this is not noted and given its due weight, the other findings, dullness, diminished breath-sounds and fremitus, almost invariably will be interpreted incorrectly as fluid or post-operative pneumonia.

Chapter VI

PERCUSSION

Dullness at the apices.
Flatness at the apex.
Flatness at the base.
Obliteration of Traube's tympany.
Tympany over a small area.
Tympany over an extensive area.
Amphoric resonance.
Hyper-resonance.
Percussion of the back.
Demonstration of the diaphragmatic excursion.
Grocco's triangle.

First of all, always compare symmetrical points on the chest, remembering that the right top normally shows impairment of resonance. Carefully mark out with a skin-pencil, Krönig's isthmuses in the supra-clavicular spaces. Note if either or both are shrunk. Such a finding results from the retraction of lung parenchyma which is associated with tuberculosis. Decide whether the dullness at the right apex is of normal or more than normal intensity. This is always more difficult to determine than dullness at the left apex, which is always pathological. If the dullness at the right apex is associated with narrowing of Krönig's isthmus, it is pathological. Having decided that one area of the chest is dull as compared with the corresponding area on the other side, percuss that area

locally and determine its boundaries, marking them with a pencil. The lower limit of apical dullness generally is determined more easily by percussion from below upward than from above downward. Slight grades of dullness at the apex also are recognized more easily by this method. Note whether the area of dullness is sharply demarcated or whether it tends to peter out at the periphery, sug-

FIGURE 1.

gesting a corresponding petering out of the underlying pathology. Note especially whether the area of dullness corresponds to some definite anatomical entity, such as one of the lobes or one of the interlobar fissures. If the former, think of the pathology that may have lobar distribution (lobar pneumonia, pneumonic phthisis). If the latter, think of interlobar effusion or empyema. Keep your mind open, however, until the examination is complete. Recall what you found when testing for tactile fremitus. Dullness, associated with normal or increased

tactile fremitus, suggests infiltration, consolidation or cavity. Dullness associated with decreased or absent tactile fremitus suggests thickened pleura, effusion, or a cavity surrounded by marked pleural thickening. The greater the dullness and the less the fremitus, the more apt it is to be fluid than thickened pleura. If pleural effusion is present, careful percussion often will show the upper

FIGURE 2. Compare the two depressions between the trachea and the lower end of the sterno-mastoid muscles, depicted in the diagram by shading. Note if one depression is more filled in and shows less shadow than the other. If so, the trachea is displaced *toward* that side. The depression away from which the trachea is displaced will look more scooped out and show more shadow.

border to follow a curved line, lowest anteriorly and posteriorly and highest in the axilla, like an enormous meniscus. In hydro-pneumo-thorax, however, the fluid seeks its own level, and the corresponding upper limit of dullness will be horizontal and will shift readily with change in position. In pleurisy with effusion, change in position causes little if any shift of dullness. Dullness at the top,

which peters out by imperceptible gradations in a downward direction into resonance, is what we are very apt to find in tuberculosis, because the tuberculous involvement tends to peter out from above downward in like manner (Fig. 3).

Flatness at an apex suggests an old cavity, because such a cavity generally has a very thick fibrous wall, which

FIGURE 3. Dullness of apical tuberculosis, petering out from above downward into normal resonance. This finding, by itself, is not enough on which to establish a diagnosis.

gives a flat percussion note. A suggestive confirmatory finding will be the pulling of the trachea over toward the side of the lesion (Fig. 4).

Flatness at the base (Fig. 5) suggests pleural effusion. Recall what you found under inspection and palpation. The combination of diminished respiratory excursion, diminished or absent tactile fremitus and marked dullness or flatness at the base means fluid or very thick pleura. These findings are easier to make over the back of the

chest. With such findings, displacement of the heart away from the side of the lesion is confirmatory of fluid.

Obliteration of Traube's tympanitic semilunar space suggests left-sided pleural effusion or massive pericardial

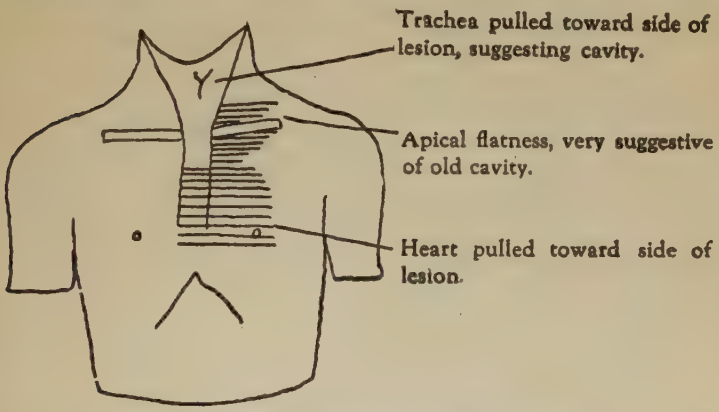
Trachea pulled toward side of lesion, suggesting cavity.
Apical flatness, very suggestive of old cavity.
Heart pulled toward side of lesion.

FIGURE 4. Flatness at an Apex.

Inspection	1. Diminished respiratory excursion on side of lesion. 2. Trachea and apex-beat pulled *toward* side of lesion.
Palpation	1. Diminished respiratory excursion on side of lesion. 2. Tactile fremitus may be unchanged or increased, but will probably be diminished as result of marked fibrosis.
Percussion	Flatness at the apex, petering out into dullness and then into normal resonance.

Diagnosis of cavity is established on the above findings.

effusion. A very high degree of flatness is found over extensive lung tumor.

Tympany is a high-pitched, drum-like, non-musical note. It can be imitated by percussing the inflated cheek, holding the lips tightly pursed. It occurs in the normal chest over Traube's space, where it is due to air in the stomach. Elsewhere it is always pathological, with one

exception, namely, that unduly hard percussion over a normal apex may elicit tympany from the large bronchi.

If tympany is localized to a small area of the chest, generally it will be at one of the apices, and, incidentally,

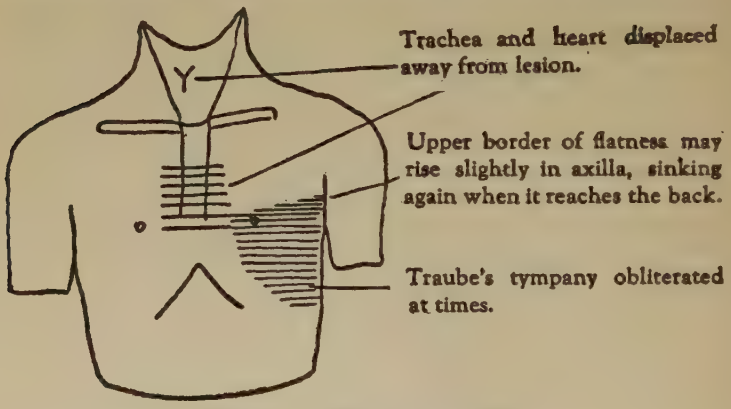
Trachea and heart displaced away from lesion.
Upper border of flatness may rise slightly in axilla, sinking again when it reaches the back.
Traube's tympany obliterated at times.

FIGURE 5. Flatness at the Base.

Inspection
1. Diminished to absent respiratory excursion on side of lesion.
2. Trachea and apex-beat pushed away from side of lesion. Apex-beat may not be visible.

Palpation
1. Diminished to absent respiratory excursion on side of lesion.
2. Diminished to absent tactile fremitus over base on side of lesion.

Percussion
1. Flatness over base on side of lesion, the upper border of the flatness perhaps rising slightly toward the axilla.
2. Traube's tympany obliterated at times in left-sided effusion.

Diagnosis of pleural effusion is established on the above findings.

generally is not recognized. Localized at an apex, it means cavity. Cast your mind back over what you have discovered under inspection and palpation. Appropriate

coincidental signs would be deflection of the trachea toward the side of the lesion, diminished expansion on the side of the lesion, increased (or decreased) tactile fremitus over the lesion, displacement of the heart toward the side

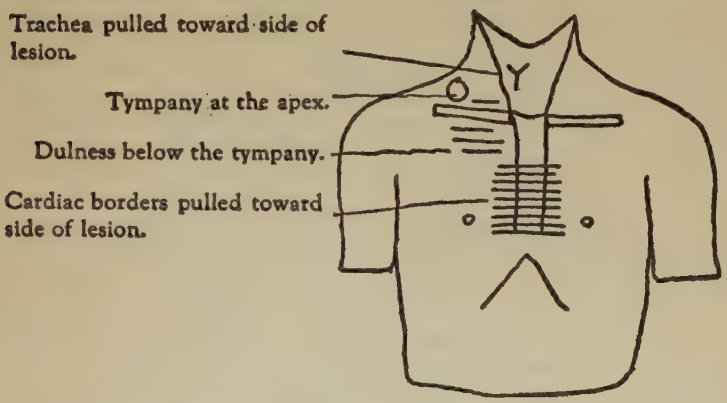
Trachea pulled toward side of lesion.
Tympany at the apex.
Dulness below the tympany.
Cardiac borders pulled toward side of lesion.

FIGURE 6. Tympany at the Apex.

Inspection	1. Diminished respiratory excursion on side of lesion. 2. Trachea and apex-beat pulled toward side of lesion.
Palpation	1. Diminished respiratory excursion on side of lesion. 2. Tactile fremitus may be unchanged or increased, but will probably be diminished as a result of marked fibrosis.
Percussion	Tympany at apex, with dullness below shading into normal resonance.

Diagnosis of cavity is established on the above findings.

of the lesion (Fig. 6). The tactile fremitus gives little or no information in this situation.

If, however, there is extensive tympany on one side of the chest, reaching from apex to base, then an entirely different situation probably is present (Fig. 7). Again, review what you have already found. Inspection probably

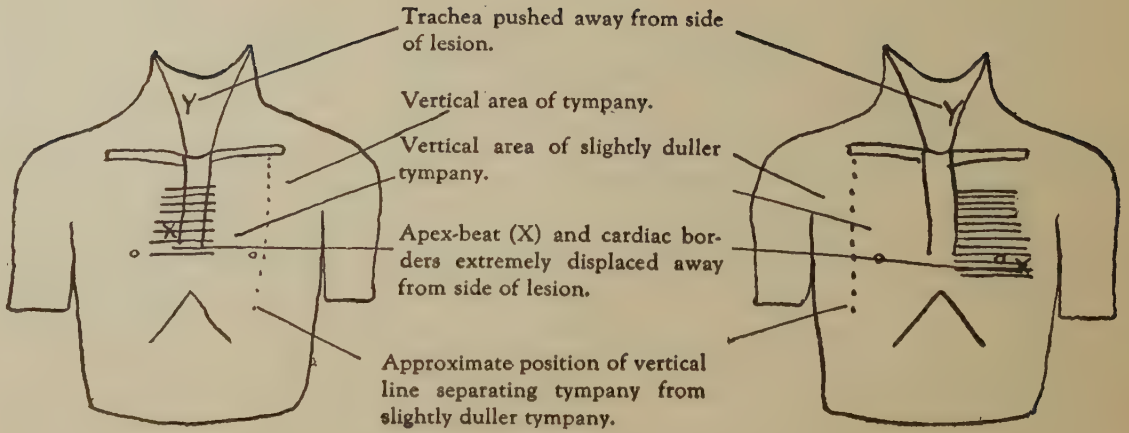
Trachea pushed away from side of lesion.
Vertical area of tympany.
Vertical area of slightly duller tympany.
Apex-beat (X) and cardiac borders extremely displaced away from side of lesion.
Approximate position of vertical line separating tympany from slightly duller tympany.
Y
X

FIGURE 7.

FIGURE 7. Extensive Tympany on One Side of the Chest from Apex to Base.

History Catastrophic onset, pain in chest, extreme dyspnœa, shock and collapse.

Inspection
1. Patient obviously critically ill.
2. Absent respiratory excursion on side of lesion.
3. Exaggeration (relative and absolute) of respiratory excursion on other side.
4. Extreme displacement of trachea and heart *away from* side of lesion. If lesion is on the left, the apex-beat may be displaced to the right of the sternum, resembling situs inversus.

Palpation Confirms 2, 3, and 4 under Inspection.
Tactile fremitus absent on side of lesion. Patient probably too ill to co-operate.

Percussion Tympany over practically all of involved side of chest. The outer half of the involved side shows pure tympany, the inner half shows slightly duller tympany, the two areas being separated by a fairly well demonstrable vertical line of demarcation, generally somewhere near the mid-clavicular line. This line of demarcation corresponds approximately to the border of the collapsed lung. Percussion confirms extreme displacement of the heart away from the side of the lesion.

Diagnosis of pneumo-thorax is established on the above findings. N. B. The crucial diagnostic finding is the displacement of the mediastinum *away from* the side of the lesion.

will have shown the patient to be in severe shock and intensely dyspnœic. The involved side will show diminished or absent respiratory movement. The other side will show respiratory movement greatly exaggerated, both relatively and absolutely. Heart and trachea will be displaced markedly away from the tympanitic side. If the lesion is on the left, the left cardiac border and apex-beat may be displaced almost to the sternum, or even beyond. Over the tympanitic side, tactile fremitus will be greatly diminished or absent, if the patient is in condition to co-operate. The final confirmatory finding will be reached under the head of auscultation. The breath-sounds will be decreased, absent, or, rarely, amphoric. This combination of findings means pneumo-thorax. Horrible to relate, frequently it is diagnosed as pneumonia. Suspecting pneumo-thorax, now carefully percuss the involved side from the axilla toward the sternum, marking the skin where a change in note occurs, not a change from tympany to dullness, but a change from tympany to less marked tympany. These marks should fall in a roughly vertical line, about in the position of the mid-clavicular line. Whenever the points representing change in percussion-note fall uniformly into an approximately geometrical line, it always arouses the suspicion of something anatomical corresponding to it. What approximately corresponds to this particular line is the border of the collapsed lung. Between this line and the sternum there is still a considerable degree of tympany, because between the collapsed lung and the chest-wall in

this area there is still a layer of air. It is only when you come to the heart that actual dullness is found, and this may not occur until the sternum is reached, so remarkably

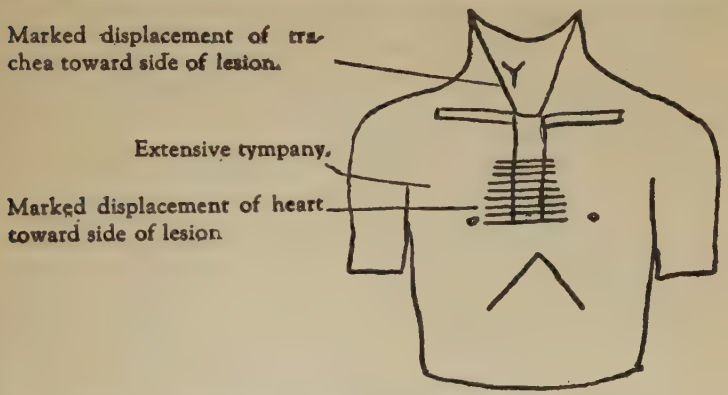
Marked displacement of trachea toward side of lesion.
Extensive tympany.
Marked displacement of heart toward side of lesion

FIGURE 8. Extensive Tympany on One Side of the Chest from Apex to Base.

History	Long, chronic illness. No acute catastrophic onset.
Inspection	1. Markedly diminished or absent respiratory excursion on side of lesion. 2. Marked displacement of trachea and heart toward side of lesion.
Palpation	Confirms 1 and 2 of Inspection. Tactile fremitus normal, increased, diminished or absent, usually the last, as result of extreme fibrosis.
Percussion	Extensive tympany over half of the chest.

Diagnosis of enormous cavity is established on these findings. Note the correspondence on the findings, with one exception, to those occurring in pneumo-thorax. The crucial differential point is the displacement of the mediastinum *toward* the side of the lesion.

may the heart be displaced. Here again we are reminded very forcibly of the vital importance of examining the heart very early in the game.

I have said that extensive tympany on one side of the

chest generally turns out to be due to pneumo-thorax. At times, but infrequently, almost an entire lobe may be involved in cavitation extending through and through the lung with practically no parenchyma left. A correspondingly extensive area of the chest-wall then may show tympany, associated with diminished respiratory movement, diminished or absent tactile fremitus and possibly with diminished or absent breath-sounds. In such a case confusion well may arise, and the erroneous diagnosis of pneumo-thorax be made. In the latter condition distress and dyspnœa are more obvious, and the mediastinum is pushed toward the opposite side.

The diagnosis is made on finding the trachea pulled toward the side of the lesion, for that is what a cavity of such extensiveness is almost certain to do. The absence of distress does not rule out pneumo-thorax, because in artificial, therapeutic pneumo-thorax the patient generally is quite comfortable (Fig. 8).

Amphoric resonance has a metallic, drum-like ring. It may be imitated by percussing the tensed cheek with the mouth held open. It is never found in the normal chest. Pathologically, it occurs in the same conditions as does tympany, and whatever has been said under the latter heading applies here. Its correlations with the findings under inspection, palpation, etc., are the same as in the case of tympany. It occurs less frequently than the latter, however.

Hyper-resonance is an exceedingly poor term. It sug-

gests an actually greater amount of resonance, a longer lasting, more resounding note, in other words, more or less of a loud pedal effect. Such is not the case at all. So-called hyper-resonance is a short, somewhat high-pitched note with a tense, boardy quality, resonance to be sure, but the almost staccato resonance of over-inflation.

As it pertains to a generalized process, emphysema, it is found over the entire lung area except parts which may be involved in localized pathology. If the chest is hyper-resonant, check back over your findings under inspection and palpation. Characteristically the correlated findings are:

1. A more or less barrel-shaped chest with the antero-posterior diameter disproportionately great as compared with the the lateral,
2. An obtuse costal angle,
3. The ribs are nearer the horizontal than is normal; the chest is in the inspiratory position,
4. Expansion is poor, and the chest moves *en masse,* i.e., the ribs move practically in unison, and not like an opening fan successively from above downward as in the normal elastic chest.

In the hyper-resonant chest, areas of dullness are hard to find, and when found are hard to demarcate accurately. It is very difficult to percuss the heart-borders with any degree of sureness. In such a chest, the auscultation of localized lesions is also difficult. Localized lesions, such as tuberculosis, are easily masked, and having established

the existence of emphysema, you will have to be more than usually alert not to miss them.

Percussion of the back follows the same general rules laid down above, but has a few special features of its own. From the apex to the angle of the scapula posteriorly, the percussion-note is less resonant than in front, because of the anatomical peculiarities, thick muscles, the angles of the ribs and the scapulæ. Light percussion and careful comparison of symmetrical points are in order. The scapula may be moved partly out of the way by having the patient put his hand on his opposite shoulder, and then raise his elbow out forward. Below the angle of the scapula the percussion-note is very resonant.

Always percuss out the lower margin of the lungs, the patient breathing very quietly, and draw a line marking the transition from resonance to dullness. Check by auscultating immediately above and below this line. The breath-sounds will be audible above, practically inaudible below. Now have the patient draw in a full breath and hold it, while again you percuss and draw a line. This line will be considerably below the first. Now have the patient breath out as far as he can and hold it, while you percuss and draw. This line will be above the first. The space between the uppermost and lowermost lines corresponds fairly well to the excursion of the diaphragm. You will be surprised how considerable it is. Watch the diaphragmatic excursion through the fluoroscope. You will see nothing more than you have demonstrated already by the humble method of percussion. If the space between

the uppermost and lowermost lines is less on one side than on the other, i.e., if the diaphragmatic motion is limited on that side, look for disease in that half of the chest. Remember that the right base is normally demonstrably higher than the left, owing to the presence of the liver (Fig. 9).

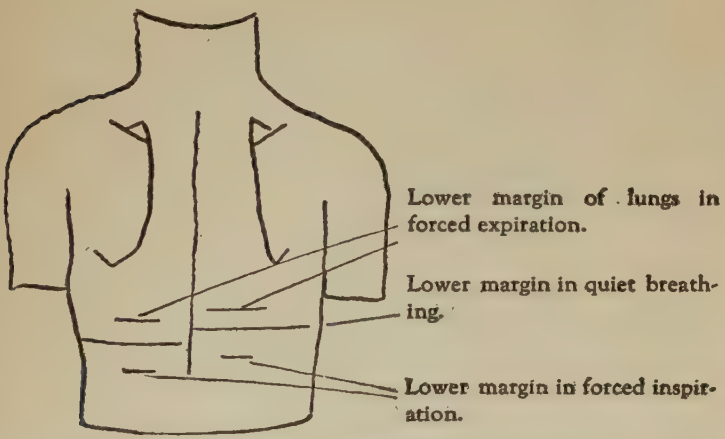

FIGURE 9. Demonstration by Percussion of the Excursion of the Diaphragm. Note that, owing to the presence of the liver, the lower margin of the right lung is always a little higher than that of the left.

Grocco's Triangle. — If you have demonstrated dullness at one base, percuss out the lower margin of the opposite lung and mark it. Then carefully percuss toward the angle between the mid-line and the lower margin of the normal lung, percussing from without inwards, and then checking by percussion from above downwards. If a triangle of dullness is found filling this angle, it is known as Grocco's triangle and is diagnostic of fluid in the oppo-

site side, being due to pushing over of the lowermost part of the mediastinum. It is a valuable diagnostic distinction between fluid and consolidation (Fig. 10).

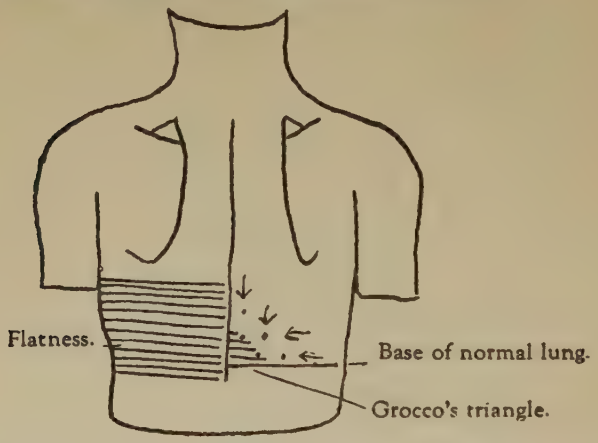

FIGURE 10. Grocco's Triangle of Paravertebral Dullness. Arrows and dots show direction of percussion. The dots represent the spots to percuss. They are not rales.

Chapter VII

AUSCULTATION

Technic.
Comparison of the apices.
The whispered voice.
Normal breath-sounds.
Exaggerated breath-sounds. Compensatory for contralateral disease.
Diminished breath-sounds.

Technic

In auscultating the chest be sure that the patient is comfortable, and that his muscles are relaxed. Tense muscles are apt to cause muscle-sounds. Always have the patient masked. Have him breathe in and out with his mouth open, slightly more deeply than usual, with a moderately quick intake and output, in a sort of gasping way, but with no increase in the respiratory rate, and always with the same intensity. The rate should be slow enough for you to be able to shift your stethoscope comfortably and unhurriedly in time to catch the next breath. Although the inspiration must be slightly forced, the expiration must not be. In expiration the chest is simply allowed to collapse passively without any muscular effort whatever. Furthermore, expiration must follow inspiration immediately. Some patients have an annoying habit of holding the breath for a perceptible interval after in-

spiration. This is not only distracting, but makes it difficult to estimate the expiratory sound correctly. Be sure that no extraneous sound is produced in the mouth or throat by air vibrating against tense or too closely held lips, against the teeth, the tongue, or the soft palate. Have the patient clear his throat and do all his swallowing before you start. It is very annoying to have the patient swallow saliva during auscultation. Very often it makes a sound like rales. Some patients, either through stupidity or through innate contrariness, never can learn to breathe the way you want. Sometimes they have stiff, muscle-bound chests. Very often they are trying too hard to help you. They feel that they have to do something and hold their bodies too stiffly erect, breathe too harshly, shrug their shoulders, or make noises in their mouths and throats. They cannot grasp the fact that you want nothing but passivity plus a slightly sharper inspiration. Show them what you want, and they will always try to improve on it. You simply will have to make the best of it.

Comparison of Apices

Now, starting in the supra-clavicular spaces, auscultate successively symmetrical points on the entire anterior and lateral chest, comparing them with respect to the four points mentioned in the first section, intensity, length of expiratory sound, pitch of inspiratory sound and quality. Pay particular attention to the supra- and infra-clavicular regions and the apices of the axillæ. The normal differ-

ences between the two sides you are familiar with. Have these questions constantly in mind:

A. Are the normal apical differences in this particular chest exaggerated in degree?

B. Are the apical differences reversed from the normal?

C. Are abnormal sounds heard in the middle and lower parts of the chest?

This last question is easy to answer and requires no discussion. The first question is harder to answer than the second. For example, you have found, as you always do, normally, broncho-vesicular breath-sounds at the right apex. It may be either normal or abnormal. Ask yourself this question: "Are these broncho-vesicular breath-sounds which I hear at the right apex of normal degree, or are they too close to the bronchial? Is the difference between the right and left apex more marked than it ought to be?" There are three legitimate answers, namely:

1. The broncho-vesicular breath-sounds at the right apex are of normal degree;
2. They are of more than normal degree;
3. I do not know.

The last may be a perfectly correct answer, and the sooner you acquire judicious ignorance the sooner are you on the path to wisdom. Human vanity, however, may take curious forms. It may express itself in false humility just as well as in pomp of the flesh and cocksureness of spirit. When you say you do not know, be sure that the circumstances are not those in which you very well ought

to know. If you are in doubt about the relative bronchiality at the right apex, refer back to what you found under percussion and ask yourself whether the right apex was duller than its normal preponderance of dullness entitled it to be. Particularly note the condition of Krönig's isthmus. If the right apex is duller than it ought to be, if its Krönig's isthmus is shrunken as compared to the left, then the right apical broncho-vesicular breath-sounds about which you are in doubt, are more apt to be of abnormal degree. Beware, however, of emaciated elderly patients. Here you are apt to find exaggerated bronchiality at the apices without any underlying pathology.

Under question "A" comes another question regarding normal degree of difference. The breath-sounds at the left apex are normally less intense than those at the right. You have got to decide:

1. Whether the intensity at the left apex is simply relatively less, i.e., in a normal degree,
2. Whether it is absolutely less, i.e., in an abnormal degree.

Again there are three possible answers, and again percussion may give the clue. Definite dullness at the left apex is absolutely abnormal and suggests that the diminished breath-sounds at that area are not merely relatively, but are absolutely and abnormally of too little intensity (Fig. 11).

Question "B" is easier to answer, i.e., are the differences between the two apices reversed from the normal, for here we have an absolute, not a relative, basis of com-

parison. Increased bronchiality and increased intensity at the left apex, as compared with the right, are definitely abnormal and often can be checked by a corresponding increase in dullness.

To sum up: Dullness at the left apex is of more significance than dullness at the right. Increased intensity of breath-sounds at the left apex is of more significance than at the right. Bronchiality at the left apex is of more significance than at the right. Decreased intensity of breath-sounds at the right apex is of more significance than decreased intensity at the left.

At this point, another inconvenient question obtrudes itself. Assume that you have found the breath-sounds at the left apex of greater intensity, i.e., louder on the left than on the right. This involves two possibilities. Either the breath-sounds are absolutely increased on the left, or they are absolutely decreased on the right. The question is by no means purely academic, as either absolute increase or absolute decrease of intensity is consistent with a tuberculous lesion, as indeed are dullness and bronchiality. The decision as to which is which must be based on a synthesis of all the various data.

Whispered Voice

A most important manœuver over an area of diminished breath-sounds, is the auscultation of the whispered voice which, you will remember, is simply a reinforced expiratory sound. Clearly enunciated whisper, or pec-

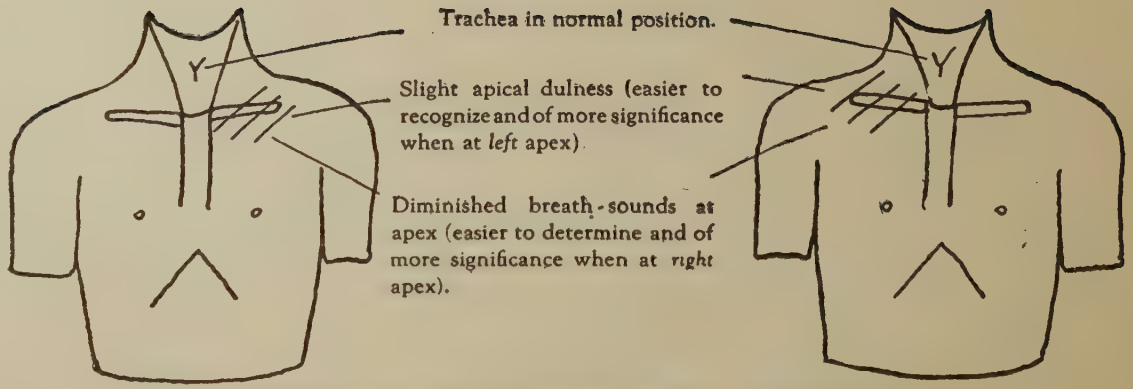
Trachea in normal position.
Slight apical dulness (easier to recognize and of more significance when at *left* apex).
Diminished breath-sounds at apex (easier to determine and of more significance when at *right* apex).

FIGURE 11. Dullness and Diminished Intensity of Breath-Sounds at Either Apex.

toriloquy, is the equivalent of a bronchial breath-sound, and, occurring over an area where the breath-sounds are so diminished as to be unidentifiable, if not actually inaudible, will betray the existence of pathology corresponding to bronchial breathing, namely, marked infiltration, consolidation or cavity. Over areas where there are frank broncho-vesicular or bronchial breath-sounds, increased whisper will be found in the former case and true pectoriloquy in the latter, with one important exception, and that a very fortunate one. At times over the apices of a very emaciated patient, you may hear breath-sounds which are almost bronchial and yet without any underlying pulmonary pathology. In such cases *you will not get the clear-cut increase in the whispered voice which you might be led to expect,* but only the degree of increase normally found at the apices, i.e., slight, or possibly, especially on the left side, no increase in whisper at all. If, however, these almost bronchial apical breath-sounds are such as a result of pathology and not merely because the chest is thin, then you will get your pectoriloquy according to Hoyle. The same differentiation between pathological and non-pathological broncho-vesicular breath-sounds at times may be necessary over the lower half of the left hilum and likewise can be determined by the presence or absence of pectoriloquy. Pneumonia of the left lower lobe may be wrongly imagined present, if this is not remembered, for over the large bronchi in this location broncho-vesicular breath-sounds sometimes are heard normally.

Auscultate with particular care all areas with abnormal palpation or percussion findings. These areas should have been marked out with a skin-pencil. They are particularly apt to show abnormal auscultatory signs. It is a good plan to compare the whispered voice in symmetrical areas over the entire chest. Increased whisper over any place but the apices and the main bronchi is abnormal.

Diagnosis should not be allowed to rest on any one finding, but should be the resultant of all findings. Some of the individual findings are of a high degree of significance. For example, dullness and broncho-vesicular breath-sounds at the left apex are not merely consistent with tuberculosis, but almost certainly mean tuberculosis. However, it is not sufficient merely to diagnose the existence of tuberculosis. The extent and pathological nature of the tuberculous lesion must be determined also, for on this depends prognosis. For this we need a synthesis of all available data, including history and x-ray findings. Sometimes even these are not enough.

This discussion of the possible percussive and auscultatory differences at the two apices has taken considerable time, for the simple reason that the permutations and combinations in an interpretative sense are somewhat numerous. In actual examination, however, a very few minutes are needed.

We will now take up individually the various auscultatory findings which may turn up and attempt to correlate them with what has gone before. As you see, the process of correlation is becoming more involved by a sort

of geometrical progression, but it is exactly what should go on in your mind during the examination of a chest, and the actual application is much more expeditious than the description.

A. Normal breath-sounds.

B. Exaggerated breath-sounds.

C. Diminished breath-sounds.

A. Normal Breath-Sounds

Normal breath-sounds are heard over normal lungs and may be heard over lungs that are considerably involved with disease. Normal breath-sounds do not rule out pathology by any means.

B. Exaggerated Breath-Sounds

Exaggerated breath-sounds are characterized by greater loudness, and the expiratory element may be more in evidence than usual. This latter circumstance may cause it to be mistaken for bronchial breathing, particularly if the breath-sounds on the other side of the chest are diminished as the result of disease on that side. In this case, it is simply compensatory for the lesser aeration of the diseased side. In such one-sided exaggeration of the breath-sounds, the exaggeration obtains over the entire side of the chest, and the breath-sounds on the opposite side of the chest are diminished, absolutely as well as relatively. It is on the latter side that we look for disease. It is quite possible, particularly in children, after cor-

rectly making a diagnosis of pneumonia by inspection, to place the pneumonia on the wrong side of the chest. This is what happens. You see the typical picture of pneumonia, high fever, flushed face, rapid pulse, herpes labialis, dyspnœa, reversed respiratory rhythm and expiratory grunt. There are no abnormal percussion findings. On one side the breath-sounds are diminished, perhaps even hard to hear. On the other side they are very loud, and the expiratory sound is prolonged and easy to hear. You then proceed to mistake the increased loudness for heightened pitch, which you have no business to do, and you fail to recognize the prolonged expiratory sound as merely the result of compensatory exaggeration. You call it bronchial breathing, and, lo and behold, there is your pneumonia on the wrong side. What profiteth it a man to diagnose pneumonia, if he places it in the wrong lung? The acquirement of merit does not lie that way. From the patient's viewpoint, it may not be a very serious error, although possibly an embarrassing one for yourself to find an alibi in case you are called for a show-down. But, from the standpoint of right and wrong thinking, the error is atrocious. A little care in inspection, noting that diminished respiratory excursion was on the same side as the diminished breath-sounds, plus a slight sense of pathological values, would have kept you straight. The real bronchial breath-sounds, which you expect to find over pneumonia, are in the process of development, and may not show up for another twenty-four hours. Again, in normal children and not infrequently in thin adult

normal chests we get universal, exaggerated breath-sounds, known as puerile breathing, with increased intensity and considerable prolongation of the expiratory sound. This can be recognized as a perfectly normal finding first by realizing that it exists, and second by noting that it is of low pitch and vesicular quality, and lastly that it is not accompanied by any increase in the whispered voice.

When, on the other hand, the breath-sounds are exaggerated, not over an entire side but over a localized area, it suggests, but does not prove, the presence of some corresponding localized pathology, infiltration, consolidation, fibrosis or cavity. Decision in such an event depends on the concomitance of other signs, change in quality or pitch, rales, or dullness to percussion. Of all physical signs, exaggeration of the breath-sounds is the least reliable, not only as regards the determination of its actual presence, but as regards the interpretation of its significance.

C. Diminished Breath-Sounds

When, however, we come to diminution of the intensity of the breath-sounds, then we are dealing with something of much greater import. Strange to say, students have a great way of fighting shy of this sign. They seem to have an inborn aversion against recognizing it. When they run across it, they often seem to think that they are simply having a harder time than usual with their ears or their

stethoscopes. This sign is by no means diagnostic of the presence of disease, but it is always suggestive. Rarely, I have found it in chests where no pathology could be demonstrated. As a suspicious sound to a vigilant sentry, this sign always should arouse the mental cry of, "who goes there?" So important is diminution of the breath-sounds, that I will devote an entire chapter to its discussion. With the possible exception of rales, it is the sign of signs. Diminution of breath-sounds almost invariably means that there is pathology of some sort or description, and yet time and again it escapes recognition, apparently solely because it is less obtrusive, and because we seem to feel that abnormal auscultatory findings should be played on the brasses and not on the wood-winds. It is because of this superlative importance of diminution of the breath-sounds, that this book's introductory chapter insists on learning how to recognize varying degrees of intensity as they obtain in the normal chest.

Chapter VIII

DIMINISHED INTENSITY OF BREATH-SOUNDS

I. Diminished Intensity over a Considerable Part of One Side of the Chest.
  A. The area of diminished breath-sounds is resonant.
    1. The patient is not in pain and is not dyspnœic. Apical tuberculosis. Artificial pneumo-thorax. Cavity. Hemiplegia.
    2. The patient is in pain and is not dyspnœic. Intercostal fibrositis. Pleurisy.
    3. The patient is dyspnœic and may or may not be in pain.
      a. Displacement of mediastinum away from side of lesion. Pneumo-thorax.
      b. No displacement of mediastinum. Pneumonia. Pulmonary embolism.
  B. The area of diminished breath-sounds is dull.
    1. The area involved is at the base.
      a. Displacement of mediastinum away from side of lesion. Effusion. Empyema. Hydro-pneumo-thorax.
      b. Displacement of mediastinum toward side of lesion. Adhesive pleurisy. Cavity. Abscess. Bronchiectasis. Massive collapse.
      c. No displacement of mediastinum. Effusion. Fibrosis. Neoplasm.
    2. The area involved is at the top of the chest. Tuberculosis. Upper lobe pneumonia. Neoplasm.

II. Diminished Breath-Sounds over a Small Area. (Apex, Base, Intermediate area). Tuberculosis. Encapsulated Fluid. Thick Pleura. Abscess.

I will discuss diminished intensity of breath-sounds under two heads:

I. Diminution over a considerable area on one side of the chest,

II. Diminution over a small area.

Having determined the presence of diminution of the breath-sounds, never, under any circumstances, fail to test the involved side for the whispered voice. Remember that the whisper is a reinforced expiratory sound, and if any hidden change in quality is present, may make it recognizable. Time and again it will bring out an unsuspected bronchial quality and thus give you the direct clue to the solution of your problem.

We will assume that the breath-sounds have been found to be diminished in intensity over a considerable part or practically all of one side of the chest. This finding enjoins on us a definite mental process, both with respect to data already acquired and with respect to data in expectancy. The various situations are handled according to the following scheme:

I. Diminution of breath-sounds over a considerable part or practically all of one side of the chest,

A. The area of diminished breath-sounds is resonant,

B. The area of diminished breath-sounds is dull.

I, A. The area of diminished breath-sounds is resonant,

1. The patient is not in pain and is not dyspnœic,

2. The patient is in pain and is not dyspnœic,

3. The patient is dyspnœic and may or may not be in pain.

(a) The heart is displaced,

(b) The heart is not displaced.

I, A, 1. *The patient is not in pain and is not dyspnœic.*

The unilateral diminution of the breath-sounds over a considerable area of chest, which is the focus of our thoughts at present, may not be particularly marked. Inspection and palpation have shown a rather slight diminution of excursion on the affected side. The trapezius muscle may be slightly more tense than on the other side, or it may be slightly atrophic, and there may be a slightly increased depression above and below the clavicle. Tactile fremitus is normal. Percussion is normal except that there may be pathological dullness at the apex. The diminution of breath-sounds is the scent. The apex is the trail, which we must subject to the closest scrutiny. Now test the apex for the whispered voice. In spite of the diminution of the breath-sounds themselves, the whispered voice may be increased. Auscultation of the breath-sounds both with and without the aid of the cough may reveal fine rales at the apex. Even without apical rales, such a combination is highly suspicious, though of course in no sense conclusive. With rales, it is certainly tuberculous infiltration. A very small active lesion at the apex may cause diminished breath-sounds over the entire side of the chest through a viscero-motor reflex analogous to that which causes spasm and rigidity over an inflamed appendix. If there are pathological changes in quality, then the greater the degree of bronchiality and pectoriloquy, the more advanced is the transi-

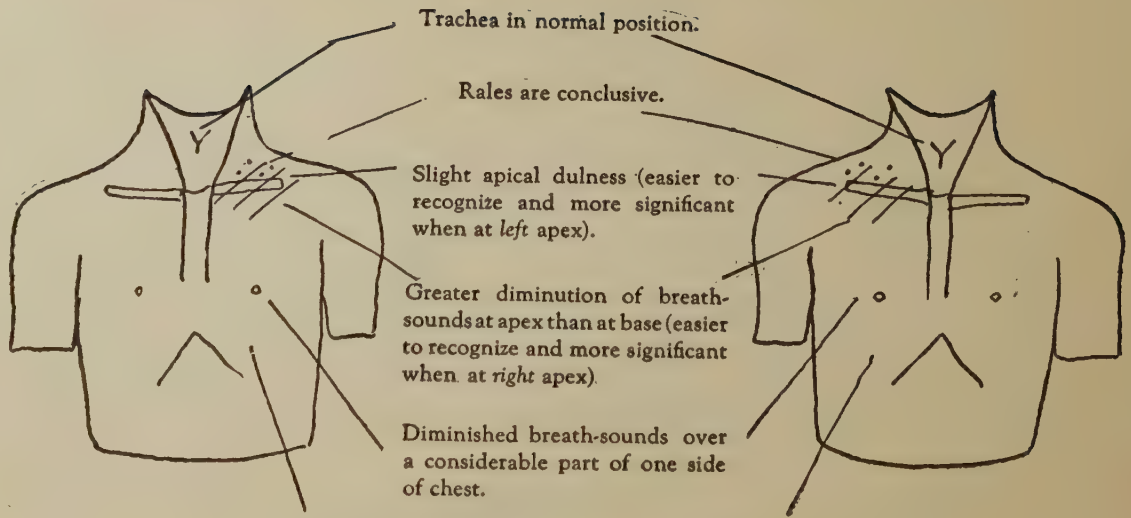
Trachea in normal position.
Rales are conclusive.
Slight apical dulness (easier to recognize and more significant when at *left* apex).
Greater diminution of breath-sounds at apex than at base (easier to recognize and more significant when at *right* apex).
Diminished breath-sounds over a considerable part of one side of chest.
Diminished respiratory excursion may be present.

FIGURE 12.

FIGURE 12. I. Diminished Breath-Sounds Over a Considerable Part of One Side.

A. The area of diminished breath-sounds is resonant (except perhaps at the apex).

1. The patient is not in pain and is not dyspnœic.

Diagnosis: Without apical rales tuberculosis is suggested. With apical rales, tuberculosis is certain.

tion from mere infiltration to consolidation or cavity (Fig. 12).

There is another condition in which you may find this combination (I, A, 1.), and this is artificial pneumothorax, or at times even spontaneous pneumothorax, the acute stage of which has passed. The first stage of spontaneous pneumothorax is accompanied almost invariably by the most profound distress or even collapse. An extensive area of resonance, which is often of a distinctly tympanitic quality, plus marked diminution or even absence of breath-sounds, means pneumothorax. If this combination turns up, refer back to what you discovered under inspection of the trachea and palpation and percussion of the heart. Displacement of the trachea and heart *away from* the side of resonance and markedly diminished breath-sounds, confirm the diagnosis of pneumothorax. As for quality, the diminished breath-sounds, if audible at all, may or may not be amphoric, and there may be pectoriloquy or amphoric whisper (Fig. 13).

There is yet another possibility. Wide-spread diminution of breath-sounds over a resonant chest, without pain or dyspnœa, plus displacement of the trachea and heart *toward* the side of the lesion, means an enormous cavity with extensive, contracted scar-tissue. In the combination under discussion, the feature diagnostic between pneumothorax and large cavity is the direction of the displacement of the mediastinum. The auscultatory findings are not conclusive, because in either case the breath-sounds and whispered voice may be diminished, absent,

cavernous or amphoric. Always look to the heart as to its position (Fig. 8).

Another condition, in which may be found the combination under discussion, is hemiplegia. In this condition there will be no displacement of the mediastinum, and the usual signs of hemiplegia will confirm the diagnosis.

To sum up our sub-division, I, A, 1, wide-spread diminution of breath-sounds over a resonant area of chest, without pain or dyspnœa, may turn out to be:

1. Early apical tuberculosis,
2. Enormous cavity,
3. Artificial pneumothorax,
4. Spontaneous pneumothorax in the stage of recovery,
5. Hemiplegia.

I, A, 2. *The patient is in pain but is not dyspnœic.*

The pain will be in the infra-axillary and infra-mammary regions, possibly in the epigastrium, and may radiate up to the shoulder and side of the neck. In a nervous patient the fright resulting from the pain may give rise to a false impression of dyspnœa. Look for tender spots in the intercostal muscles. If they are present, the diagnosis is fibrositis of the intercostal muscles, a very frequent condition, which generally is incorrectly called intercostal neuralgia, or worse, neuritis. Pleurodynia is another term wrongly attached to this condition. Fibrositis is an inflammation of fibrous tissue, the tendons and interstitial connective tissue of the muscles. It is of interest to us, when occurring in the intercostal spaces, because in this situation frequently it is mistaken for pleurisy, a condition

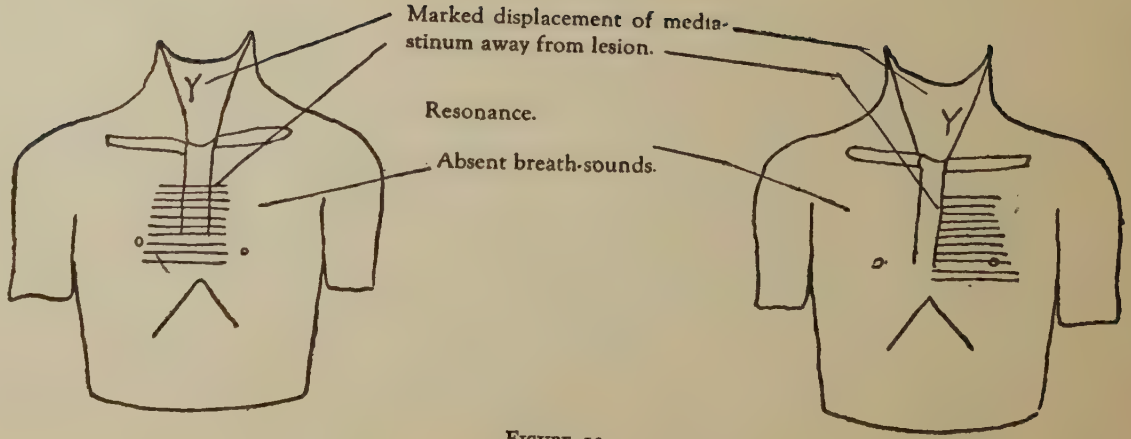
Marked displacement of mediastinum away from lesion.
Resonance.
Absent breath-sounds.

FIGURE 13.

FIGURE 13. Diminished Breath-Sounds Over a Considerable Part of One Side.

A. The area of diminished breath-sounds is resonant.

1. The patient is not in pain and is not dyspnœic.

Inspection
1. Absent respiratory excursion on side of lesion.
2. Exaggeration (relative and absolute) of respiratory excursion on other side.
3. Extreme displacement of trachea and heart *away from* side of lesion.

Palpation
Confirms 1, 2, and 3 of Inspection.
Tactile fremitus absent over side of lesion.

Percussion
Tympanitic resonance over side of lesion.
Heart markedly displaced *away from* side of lesion.

Auscultation
Absent breath-sounds over side of lesion. Exceptionally there may be very distant amphoric or cavernous breath-sounds, or amphoric whispered voice.

Diagnosis, deduced from the three crucial signs of absent breath-sounds, plus resonance, plus displacement of the mediastinum *away from* the side of the lesion, is pneumo-thorax. The absence of dyspnœa means that the pneumo-thorax is either artificial, or spontaneous after the initial period of shock.

of vastly different significance and implication. This error is particularly apt to occur in a severe case, because the pain, the grunting on too deep inspiration and the inhibition of respiratory motion may be very marked indeed. Look for markedly tender spots. If present, they practically establish the diagnosis. Pain and tenderness in parts of the body other than the chest are of helpful significance. There are no friction-sounds, but their absence does not rule out pleurisy. A friction-rub, generally heard in the lower axilla, of course means pleurisy. But here a caution is needed. The inflamed muscles of fibrositis may give rise to exaggerated muscle-sounds, which readily may be mistaken for a friction-sound. If palpable friction-rubs are present, they, of course, mean pleurisy. Constitutional symptoms do not help us to differentiate between fibrositis and pleurisy, nor does limitation of the motion of the diaphragm, to be described when we come to examine the back of the chest. The temperature may be normal or elevated, and the diaphragmatic excursion may be limited in both conditions. If the pain is very low in the axilla, or in the epigastrium, particularly if it radiates to the corresponding shoulder or side of the neck, diaphragmatic pleurisy must be considered. In this condition a pleural friction is not apt to be present, although it may make its appearance in the course of a few days. To diagnose diaphragmatic pleurisy without a friction-rub is simply to guess. The chances are that the condition really is fibrositis.

To sum up, diminution of the breath-sounds over a con-

siderable area of resonant chest, with pain but without dyspnœa, generally means one of two things:

1. Intercostal fibrositis,

2. Pleurisy.

Their differentiation is of the highest importance.

I, A, 3. *The patient is dyspnœic and may or may not be in pain.*

The patient is obviously acutely ill, possibly in collapse. The temperature is normal or elevated even to 104 degrees Fahrenheit. The pulse is very rapid. There may be pain and reversal of respiratory rhythm, sudden arrest of the inspiratory movement followed by a pause and an expiratory grunt such as may occur in fibrositis and pleurisy. Dyspnœa, however, stands pre-eminent and distress is urgent, not intermittent and contingent upon too deep an inspiration, but constant and merciless. One side of the chest is not moving as freely as the other side, is resonant to percussion and shows marked diminution or absence of breath-sounds. The other side of the chest shows exaggerated motion and a compensatory exaggeration of breath-sounds, which you must be on the alert not to mistake for bronchial breathing. First of all, look to the heart.

I, A, 3 (a). *The patient is dyspnœic and may or may not be in pain; the heart is displaced.*

If the heart and trachea are displaced away from the side of diminished breath-sounds, then you are dealing with a spontaneous pneumothorax, which, by implication, probably means tuberculosis (Fig. 7). The

next step is to give, parenthetically, a good husky dose of morphine and atropine, the morphine to quiet the distress, the atropine to cut off the terrific cardio-inhibitory stimuli, which are sweeping over the vagus, and which at any moment may bring disaster. Any further manipulation of a patient in the parlous first stage of spontaneous pneumothorax is unpermissible. Later on, if he survives and is more comfortable, the front of the chest may be examined with greater detail. But note, that the systematic mental approach which we have just described is sufficient for accurate diagnosis in this situation of intense emergency. Later, supplementary examination will disclose the following. Tactile fremitus on the diseased side, if the patient is able to co-operate sufficiently to demonstrate, is absent or markedly diminished. Careful percussion shows that the resonance of the diseased side is of a tympanitic, rarely of an amphoric, quality. Very careful percussion from the axilla toward the sternum shows a slight change in note somewhere in the neighborhood of the mid-clavicular line corresponding approximately to the border of the collapsed lung. The displacement of the heart may be extreme. A left-sided pneumothorax may push the left cardiac border over to the left sternal margin and the apex-beat over to a point midway between the right nipple and the sternum. A right-sided pneumothorax may push the right cardiac border over to the left sternal margin and the apex over to the left axilla. The breath-sounds still may be absent or may be faintly amphoric. If the patient is able to

co-operate, an amphoric whisper may be demonstrated. The coin-sound, a detestable sign, may be present. My animus against the coin-sound is for this reason: It is very exasperating to an enthusiastic and artistic instructor to give a student a case of artificial pneumothorax to diagnose, have him get a fool hunch right at the start and look for the coin-sound. Having found it, he escapes the austere but fruitful inductive discipline which alone can stimulate his observing and reasoning powers and proceeds by the intellectually degrading method of deduction to demonstrate all the signs in the catalogue, and all this with a confounded air of self-satisfaction that is to the highest degree annoying. It may be magnificent, but it does not make for the progress of the world.

If the patient recovers sufficiently to give you any kind of a history, he will tell you that, out of a clear sky, he experienced a sudden violent pain in the chest, followed immediately by dyspnœa of the most urgent and appalling kind, plus shock or even unconsciousness. Find out what he was doing when the attack came on, whether he was at comparative rest, or whether he was engaged in some violent strain. Most authorities consider that all cases of spontaneous pneumothorax, not the result of external trauma, mean tuberculosis and are caused by the rupture by intra-pulmonary air-pressure of sub-pleural tuberculous tissue into the pleural cavity. They insist that the normal visceral pleura is impervious to blow-outs from any strain that the body itself is capable of producing. There is, however, some doubt as to the 100 per cent.

validity of this rule. If the pneumothorax comes on during comparative rest, it is almost certainly of tuberculous origin. If, however, it comes on during excessive strain, such as weight-lifting, violent coughing or vomiting, then there is certainly a chance that it is non-tuberculous. I have had several cases of spontaneous pneumothorax occurring under strain, in which the most careful investigation and long-continued observation have failed to demonstrate the slightest indication of tuberculosis. The failure to demonstrate tuberculosis, however, does not rule it out infallibly, and the safe rule to follow is that every case of pneumothorax, like every case of hemoptysis or pleurisy with effusion, is tuberculous until a preponderating mass of evidence, both objective and symptomatic, rules otherwise.

I, A, 3, (b). *The patient is dyspnœic and may or may not be in pain; the heart is not displaced.*

If, on the other hand, the heart is not displaced, with diminished breath-sounds over a considerable resonant area plus dyspnœa, we are dealing with one of two things, early pneumonia or pulmonary embolism. In the former condition we find high fever, rapid bounding pulse and a flushed face. Inspection of the chest shows rapid breathing with limitation of motion on the diseased side. In the early stages probably there will be no change in tactile fremitus, no dullness and no change in the quality of the breath-sounds. The breath-sounds, however, will be diminished on the diseased side, and there will be a corresponding compen-

satory exaggeration of intensity and increase in expiratory sounds on the uninvolved side. For this reason, the pneumonia, diagnosed correctly by the general aspect and symptoms, may be mistakenly thought to be on the healthy side. In the course of a few hours or even days, there will develop dullness, bronchial breath-sounds and bronchial whisper of lobar distribution, or possibly limited to a small area. There probably will be rales also, at first fine, later, coarser.

In pulmonary embolism there may be a sudden catastrophic onset with profound collapse or even coma. The patient may die practically instantaneously from shock, or a few hours later from a swiftly developing pulmonary œdema, diagnosed by the presence of many large bubbling rales throughout both lungs, particularly in the lower lobes. The process, however, need by no means be so dramatic, and if the patient survives, as he frequently does, there may develop in the course of a day or two an area of dullness, bronchial breath-sounds, bronchial whisper and moist rales, small in extent and rather difficult to determine unless you make a special search for it. Pain is a very frequent concomitant of embolism, and fever generally is present for a few days. The probability of the diagnosis is increased by the presence of a potential source of emboli. Occasionally, a few moments after an intramuscular injection of an insoluble drug, such as mercury or bismuth salicylate, the patient may experience in mild or severe form the symptoms just described, plus a metallic taste in his mouth. Generally he survives, but he

may be very ill for a couple of weeks. Such a situation means pulmonary embolism of the injected drug. It is a disconcerting experience.

I. Diminution of breath-sounds over a considerable area on one side of the chest,

B. The area of diminished breath-sounds is dull to percussion.

Unfortunately this picture cannot be given the circumstantial classification which we used before. The possibilities are too numerous, and there is too much overlapping of signs and symptoms. The best frame of reference on which to hang our findings in the solution of "I, B," is the position of the involvement in question, i.e., whether it is in the upper or the lower part of the chest. Having established this as a point of departure, a convenient secondary division will be according to the position of the trachea and heart, whether they are normal in position, displaced away from, or displaced toward, the side of the lesion. This matter of the position of the heart always keeps bobbing up. It is impossible to lay too much emphasis upon it. If the heart has been examined at the start of the procedure, a great deal of mental effort will have been rendered unnecessary, and a considerable factor of error and lost motion eliminated. The tentative scheme of mental approach is as follows:

I. Diminution of breath-sounds over a considerable area on one side of the chest,

B. The area of diminished breath-sounds is dull to percussion,

1. The area involved is at the base,
   (a) The heart and trachea are displaced *away from* the side of the lesion,
   (b) The heart and trachea are displaced *toward* the side of the lesion,
   (c) The heart and trachea are in *normal position.*
2. The area involved is at the top of the chest.

I. B, 1 (a). *The area involved is at the base; the heart and trachea are displaced away from the side of the lesion (Fig. 14).*

The patient may or may not be dyspnœic. Inspection shows diminished excursion on the affected side. Palpation confirms this and in addition shows markedly diminished or absent tactile fremitus. The tactile fremitus may, exceptionally, fail to show this significant reduction. Percussion shows flatness or marked dullness at the base, merging into merely impaired resonance and then resonance from below upward. The upper border of dullness is not horizontal, but is slightly curved above like an enormous meniscus, the peak of which is apt to be in the lateral part of the chest. The breath-sounds are absent or markedly diminished, but may be audible, though diminished, in the upper, more resonant part of the chest. Somewhere in the neighborhood of the upper border of dullness there may or may not be a small area of diminished bronchial breath-sounds or increased whispered voice. This combination of signs means fluid in the pleural cavity. A confirmatory finding is the presence of

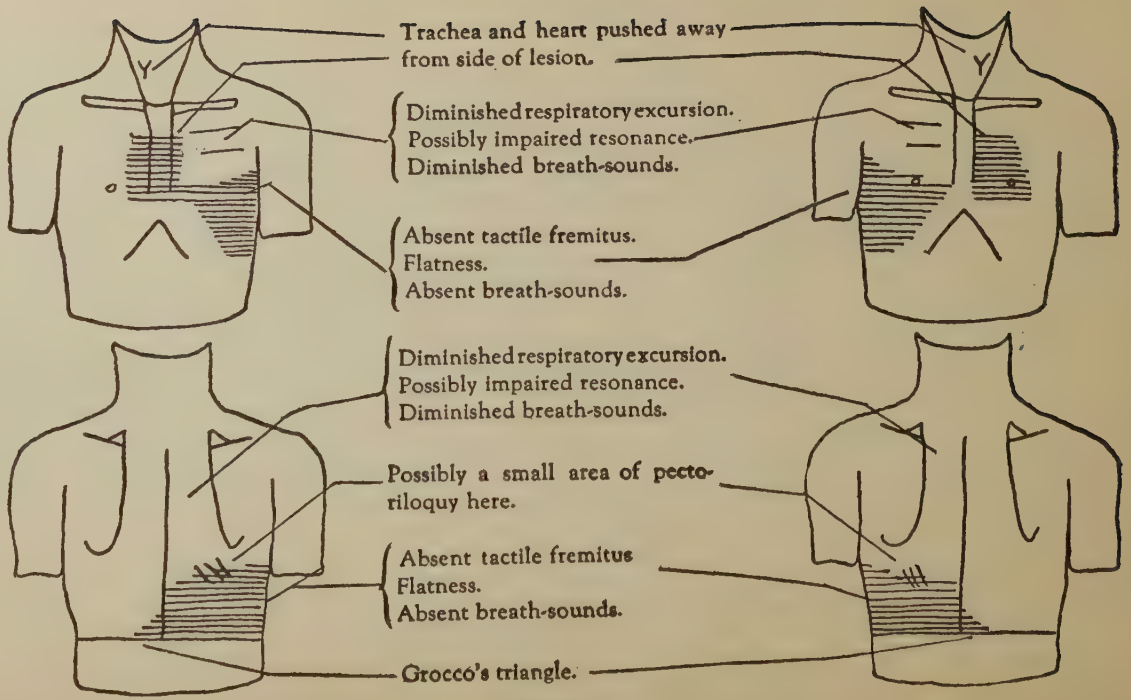
Trachea and heart pushed away from side of lesion.
Diminished respiratory excursion.
Possibly impaired resonance.
Diminished breath-sounds.
Absent tactile fremitus.
Flatness.
Absent breath-sounds.
Diminished respiratory excursion.
Possibly impaired resonance.
Diminished breath-sounds.
Possibly a small area of pectoriloquy here.
Absent tactile fremitus
Flatness.
Absent breath-sounds.
Grocco's triangle.

FIGURE 14.

FIGURE 14. I. Diminished Breath-Sounds Over a Considerable Part of One Side.

B. The area of diminished breath-sounds is dull.

1. The area involved is at the base.

(a) The heart and trachea are displaced away from the lesion.

Inspection

1. Diminished to absent respiratory excursion on side of lesion.
2. Exaggeration (relative and absolute) of excursion on other side.
3. Moderate displacement of heart and trachea *away from* side of lesion usually but not always present.

Palpation

Confirms 1, 2, and 3 of Inspection.
Tactile fremitus absent over base.

Percussion

1. Possibly impaired resonance over upper half of chest.
2. Flatness at base, the upper border of the flatness rising slightly in the axilla.
3. Grocco's triangle of dullness on opposite side.

Auscultation

1. Diminished breath-sounds over upper half of chest.
2. Absent breath-sounds over base.
3. Possibly a small area of pectoriloquy near upper level of flatness.

Diagnosis, pleurisy with effusion. The crucial diagnostic findings are absent tactile fremitus, flatness and absent breath-sounds. The signs of displacement of the mediastinum *away from* side of the lesion are confirmatory if present.

Grocco's paravertebral triangle of dullness, which is found in the angle between the spinal column and the base of the opposite lung, and is caused by displacement of the mediastinum. If the patient shows a normal or merely moderately increased pulse and temperature, the fluid is probably serous, *and this means tuberculosis in the vast majority of cases.* The books say it means tuberculosis in 80 per cent. of cases, but do not pay any attention to the books. The presence of serous effusion means tuberculosis, unless you can disprove it, and the odds are a hundred to one that you cannot. Serous effusion, with nothing else to back up your opinion, is almost as pathognomonic of tuberculosis as is the finding of bacilli in the sputum. Any attempt to side-step this inscrutable and inexorable fact springs either from ignorance or from moral and intellectual cowardice. It is a diagnostic crime that time after time has led to disaster.

If the temperature and pulse are high with marked toxemia and leukocytosis, with history of sudden onset or precedent pneumonia, look for empyema. So-called unresolved pneumonia frequently turns out to be pus in the pleural cavity.

The above combination of signs may be limited to, or more definitely obvious in, a wedge-shaped area in the neighborhood of one of the interlobar fissures. Think of interlobar fluid, either serum or pus. Or the signs may have an atypical extension, the margins not being referable to anatomic considerations. In such an event think of encysted effusion, delimited fortuitously by ad-

hesions, again signifying either serum or pus, and again still involving the differentiation between tuberculosis and surgical empyema. If your examination has shown a pneumo-thorax, be sure to look for fluid at the base. The signs over the fluid will be as just described, except that the upper level of dullness will be horizontal instead of slightly convex and will shift readily with change of position.

N.B. — In pleural effusion, particularly if interlobar, the contra-lateral displacement of the heart may be absent or so slight as to escape notice. In this event we depend for our diagnosis largely upon the diminution or absence of tactile fremitus.

I, B, 1 (b). *The area involved is at the base; the heart and trachea are displaced toward the side of the lesion (Fig. 15).*

The determining feature of this group of findings being the displacement of the heart and trachea toward the lesion, the question arises as to what is the nature of the pathological process, which is competent to accomplish such an effect. There are two processes which can do this:

(α) An extensive scar-tissue formation, which, by its gradual contraction, pulls the mediastinum over toward it,

(β) A process involving a reduction in volume of the affected lung, without the latter being separated from the chest wall. In this case, the heart and trachea are not pulled toward the lesion, but are

pushed toward it by the predominating expansive effect of the normal lung on the other side.

The first type of displacement may occur to a limited extent as the result of a very considerable chronic adhesive pleurisy which itself may be non-tuberculous in origin (history of acute fibrinous pleurisy, pneumonia or empyema). The history plays an important part in the decision. It is with cavity formation, however, that we find that very high degree of scar-tissue formation which is needed to effect a really obvious displacement of the mediastinum. Associated with the cavity formation, we generally find a considerable amount of chronic adhesive pleurisy, which may mask the signs of the underlying condition. The chronic adhesive pleurisy, *per se*, may cause:

(1) Dullness, extensive in area and more or less marked in degree depending on the thickness of the layer of scar tissue,

(2) Diminution of breath-sounds and tactile fremitus, the diminution again being proportional to the degree of fibrosis,

(3) A palpable or audible friction-rub,

(4) Diminution of the excursion of the diaphragm, described under the examination of the back,

(5) Slight or moderate displacement of the heart toward the side of the lesion.

Under this smoke-screen of signs, we search for cavity, remembering that, *if the heart is obviously displaced,* cavity of some sort probably is present. If, one to two

weeks following an operation, or following the suspicion of having inhaled a foreign body, which possibility always must be inquired into scrupulously in the case of small children and must be suspected even when the results of the inquiry are negative, if, with such precedent circumstances, the patient has begun to sicken and in the course of a few days shows high temperature, rapid pulse, leukocytosis, cough with expectoration that usually quickly becomes profuse and foul, dyspnœa and cyanosis, and you find the signs mentioned under our present heading, you are probably dealing with a pulmonary abscess, and upon your alertness and openness of mind may depend the life of your patient. Of course, at first, displacement of the heart will be in abeyance, for it is only after considerable tissue-reaction and scar-formation have set in that such displacement can occur. But, keep looking for it. The sputum may not be foul, but this must not deceive you into the conventional mistake of diagnosing the condition as pneumonia. Superficially the two diseases may resemble each other, but in abscess the onset is slightly more gradual, and the temperature is definitely septic. In pneumonia the onset tends to be more explosive and the temperature unremitting. Pneumonia, unless complicated by empyema, typically ends by crisis somewhere around the ninth day. The symptoms of abscess, on the other hand, persist. Empyema itself may be difficult to rule out, particularly in the early stages. It generally gives rise to the fairly definite signs of fluid, which, if present to any degree, is apt to displace the heart *away from* the

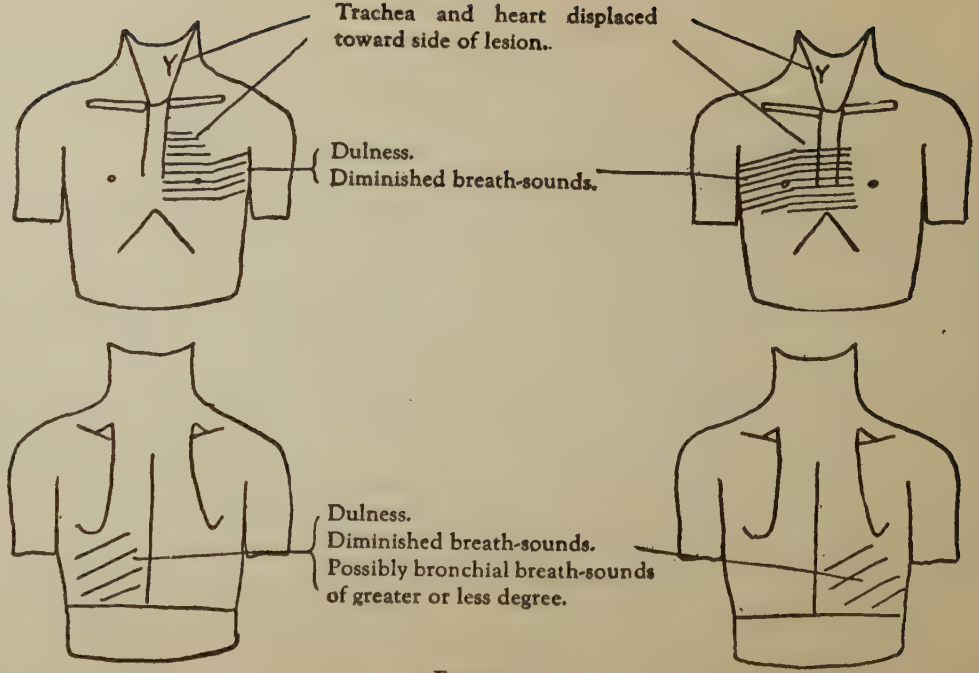
Trachea and heart displaced toward side of lesion.
Dulness.
Diminished breath-sounds.
Dulness.
Diminished breath-sounds.
Possibly bronchial breath-sounds of greater or less degree.

FIGURE 15.

FIGURE 15. *I. Diminished Breath-Sounds over a Considerable Area on One Side of the Chest.*

B. The area of diminished breath-sounds is dull to percussion.

1. The area involved is at the base.

(*b*) *The heart and trachea are displaced toward the side of the lesion.*

The above combination of findings is consistent with:

(*1*) *A very extensive fibrinous pleurisy.* (Sequel of acute fibrinous or serous pleurisy, empyema, or pneumonia.) There may be a palpable or audible friction. There are no changes in the quality of the breath-sounds. The displacement of the mediastinum, if present at all, is slight and is only present if the pleural fibrosis is marked.

(*2*) *Cavity.*

a. History of rather stormy onset about a week after operation or inhalation of foreign body. Septic fever. Cough and expectoration that soon becomes foul and profuse and separates on standing into three layers. Clubbing of the fingers almost always takes place and is established in a short time. Repeated sputum examinations invariably are negative for tuberculosis. Dullness may be slight in extent and hard to find. The only invariable physical sign is diminution of the breath-sounds, and this may be missed as a result of the usual prejudice against finding this sign. Other auscultatory signs are conspicuous by their absence, possibly a small patch of distant bronchial breath-sounds or pectoriloquy, and possibly a few localized moist rales. The heart becomes considerably displaced as the disease progresses. Physical signs, if present, will probably be more marked at the back of the chest. *Diagnosis, Pulmonary abscess.*

b. History of chronic cough, and profuse, sometimes foul, expectoration, separating into three layers. Recurrent attacks of broncho-pneumonia. Displacement of heart is considerable. Fingers generally clubbed. Auscultatory signs are much in evidence (bronchial breath-sounds and many moist rales). N. B. The breath-sounds may not be of diminished intensity, but may be louder than normal. *Diagnosis, bronchiectasis.*

c. Chronic pulmonary history. Fingers may be slightly clubbed. Auscultatory signs of cavity definite (bronchial breath-sounds and medium moist rales). Sputum positive for tuberculosis, and this is the only criterion by which the diagnosis can be made. *Diagnosis, basal tuberculosis, third stage.* Very rare without involvement of the upper lobe.

(*3*) *Reduced volume of affected lung, without separation from chest-wall.*

History of recent operation or trauma. Heart displaced *toward* lesion. Slight dullness at base. Breath-sounds diminished in intensity, possibly distantly bronchial in quality. *Diagnosis, post-operative atalectasis, or massive collapse.* Almost *always* mis-diagnosed as post-operative pneumonia. Very important to recognize, as it may progress to pneumonia and may be cured by forced inflation of the affected lung, by rolling the patient on the sound side and having him cough, or by causing hyperpnœa by administering carbon dioxide. Should always be suspected in post-operative pulmonary disease. The crucial finding is the displacement of the heart *toward* the side of the lesion.

side of the lesion. In case of doubt, use the exploring needle. The sputum is a valuable differentiating point between abscess on the one hand and pneumonia on the other. As for intra-pulmonary signs, they are usually outspoken in pneumonia and leave no doubt as to the existence of frank consolidation. In abscess the signs are generally few and far between, a small patch of medium moist rales or bronchial breath-sounds or whisper, or nothing at all. In abscess the dullness may be small in extent and slight in degree. In empyema it is marked and wide-spread. It is only when the abscess reaches a later stage that extensive and marked dullness from intra- and extra-pulmonary sources occurs, and it is only then that we can expect any obvious displacement of the heart. Clubbing of the fingers is almost constant in pulmonary abscess. It may be established with extraordinary swiftness, in even three or four weeks. It does not occur, unless very exceptionally, in empyema.

Another possible interpretation of the group of signs under discussion, is a disease of much more insidious onset, which may be characterized by recurrent attacks of broncho-pneumonia. In the intervals of comparative freedom from toxic symptoms, there are, nevertheless, manifestations of chronic pulmonary disease, cough with profuse foul expectoration, dyspnœa on slight exertion, pallor or cyanosis, under-development and, generally, marked clubbing of the fingers, although this last sign at times may be absent. The onset may be insidious and gradual, possibly dating back to a bronchitis or broncho-

pneumonia. During the acute febrile exacerbations the sputum increases in amount and foulness; it separates into three layers on standing. The intra-pulmonary signs of cavitation (bronchial breathing, cavernous breathing and medium moist rales) are much more in evidence than in the case of abscess, and the intensity of the breath-sounds is more apt to be exaggerated than diminished. Furthermore, the disease is often bilateral, in which case displacement of the heart is not in evidence. The diagnosis of this condition is bronchiectasis. The signs are those of extensive destructive and reparative pathology in one or both lower lobes, rarely in an upper lobe, with frequently repeated failure to find tubercle bacilli in the sputum. The only situation, where a negative sputum has any weight in the ruling out of tuberculosis, is where there have been numerous negative examinations *in the presence of extensive pathology.*

There is yet another and perhaps not so very rare condition causing the combination of signs under the present heading. The condition comes on a day or two after operation. Dyspnœa is more in evidence than toxemia. Tactile fremitus is diminished. The condition generally is diagnosed erroneously as effusion or post-operative pneumonia. The deciding factor in the diagnosis is the displacement of the heart *toward* the lesion. This combination of signs and symptoms means massive pulmonary collapse, or post-operative atalectasis. The mechanism of its cause is as follows. General anæsthesia, reinforced by morphine, abolishes the cough-reflex and tends to cause

shallow breathing with diminished ventilation. An accumulation of mucus may plug up a large bronchus, almost always at the base. The tributary area of vesicles is deprived of ventilation, the contained air becomes absorbed, the vesicles collapse, their walls adhere and a large area of atalectasis results. The involved lobe becomes reduced in volume, and the heart is pushed over toward it by the predominatingly expanded normal lung. Rolling the patient on the sound side and getting him to cough will sometimes cause a sudden clearing up of the condition. Inducing hyperpnœa by administering carbon dioxide sometimes will have the same effect.

I, B, 1 (c). *The area involved is at the base; the heart and trachea are in normal position.*

This grouping is easily summed up. It includes all the pathology grouped under "I, B, 1 (a)", and "I, B, 1 (b)". In the former instance the effusion is not large enough to displace the heart perceptibly away from the lesion. In the latter instance the fibrosis is not of sufficient degree to draw the heart toward the lesion. Each case will have to be decided on its merits, without reference to the position of the heart. Besides these, however, there are two conditions showing the above signs, in which, with one infrequent exception, it is intrinsically impossible for the heart to be shifted. Given an acute, stormy onset, with high fever, cough, possibly rusty sputum, pain in the chest, dyspnœa and leukocytosis, this combination of signs means *pneumonia.* In fact, even the dullness may be lacking, the only physical signs at the onset being dimin-

ished breath-sounds and diminished respiratory excursion. In the course of twenty-four hours a few fine rales may be heard, and after another day bronchial breath-sounds may enter the picture. The dullness and bronchial breath-sounds may be considerate enough to establish themselves over a reasonably accurate lobar distribution, but this is not apt to happen. Very frequently they are in evidence only over a small patch. The ubiquitous exception, referred to above, is that in very young children the increased compensatory ventilation of the sound lung rarely may displace the mediastinum toward the diseased side.

The second of the two conditions in which it is impossible to get displacement of the heart, is insidious in onset. The only symptoms may be an unobtrusive cough without expectoration, slight but progressive loss of weight and strength and a very gradually decreased ability to undergo effort without dyspnœa. The dullness is of very high degree, even to flatness, and may be of an easily determined lobar distribution. Diminution of breath-sounds and tactile fremitus is quite definite. There is generally no change in the quality of the breath-sounds, although increased whisper may occur. The diagnosis is almost certainly *pulmonary neoplasm.*

I, B, 2. *The area involved is at the top of the chest.* Here we may have any of the foregoing possibilities described under " I, B, 1 ", and the criteria of differentiation will be the same, including the symptom-pictures described under " I, A, 1, 2, 3 ". There will, however, almost

never be found any lesion capable of displacing the heart to the opposite side. The only lesion capable of doing this is the very unlikely and infrequent event of fluid encysted at the apex.

In the first place, the mere fact of a chronic lesion's occurring at the top instead of at the base, makes tuberculosis always the most likely candidate, just as when the lesion was limited to the base, tuberculosis was the least likely. We must, however, never lose sight of the fact that non-tuberculous lesions frequently occur at the top. An extensive lesion at the top, if tuberculous, is apt to show, but not necessarily, however, two phenomena. If the lesion is of some chronicity, there is apt to be displacement of the mediastinum toward it. Furthermore, an extensive active tuberculous lesion is apt to show, sooner or later, a positive sputum. Absence of these two findings does not rule out tuberculosis by any means, but must put us particularly on the look-out for upper lobe pneumonia. In the absence of displacement of the mediastinum and positive sputum, the probability of lobar pneumonia varies directly with the acuteness of the onset and symptoms, that of tuberculosis varying inversely. We must not forget that lobar pneumonia at times, especially in elderly people, may be unaccompanied by its typical stormy symptoms. Here is a case in point. A man of 55 was admitted to the tuberculosis ward, which in most hospitals is simply a clearing house for lung cases. He had a temperature of 101° F., a slight cough and was not especially distressed. From the right apex to the

third rib there were slight dullness, diminished breath-sounds and a few fine moist rales. There was no displacement of the mediastinum. The x-ray showed extensive involvement of the right upper lobe. Advanced tuberculosis was diagnosed. At the end of a week, he was much improved, and the attending physician was about to approve him for transfer to a tuberculosis sanitorium, when he was mildly struck by the fact that the sputum was negative, although the extent of the disease was considerable. The physical signs were about the same. A second x-ray showed a complete clearing up of the lesion. The patient did not have tuberculosis at all. He had had pneumonia. Now a mistake like this is no joke, although it has happened before and will happen again. In the first place, the patient should never have been sent for x-ray, with its attendant exposure and increased risk of spreading the disease. In the second place, it was fool luck and not good management that saved this patient from being branded as tuberculous for the rest of his life and being packed off with a " lettre de cachet " to the Bastille in the shape of a tuberculosis sanitorium. And yet, for the life of me, I do not see how this case could have been diagnosed except by the actual course of the disease. The only answer is to be super-normally alert all of the time, and, if you are working in a tuberculosis ward, not to be overwhelmed by the psychological momentum of that fact.

Another diagnostic possibility, consistent with the above group of findings, is upper lobe neoplasm. Highly sug-

gestive diagnostic points in such a case are chronicity without displacement of the mediastinum. A tuberculous lesion of considerable chronicity and extent would be especially apt to pull the mediastinum over.

I, B. *The area of diminished breath-sounds is dull; the area is small* (*apex, base, intermediate area*).

If the small area of diminished breath-sounds is at the top, tuberculosis is the most likely possibility and must be considered present until ruled out. In fact, this is one of the earliest signs of tuberculosis, although it is in no sense pathognomonic, but must be supplemented by other data such as hemoptysis or rales. It is quite obvious that the earliest suspicious sign of tuberculosis is one whose recognition must be sedulously cultivated. Nevertheless, this one particular sign-post is overlooked more than any other. None but the expert seems capable of recognizing and appraising it. And yet all that it involves is the decision as to which of two breath-sounds heard over symmetrical points on the chest is less loud. The difference in loudness will be no greater, no more subtle, no more difficult to establish, than that between the normal right and left apices. If recognition of the latter has been conscientiously acquired according to the scheme described in the introductory chapter, then this crowning and yet so easily attained subtlety of auscultation will fall within your grasp.

If the area of diminished breath-sounds is at the base or in an intermediate position, think of fluid, thickened pleura or abscess. Fluid in an intermediate area of neces-

sity would be encapsulated or interlobar. Finding such a deposit and confirming its presence with the needle gives the thrill that all true intellectual adventure grants. Pulmonary abscess, unlike bronchiectasis, is notoriously parsimonious of physical signs. Just this small area of diminished breath-sounds is the only really constant finding in this condition. Pneumonia or infarct may be present. Remember that physical examination is an inductive process. The final diagnosis is the resultant of all data that have been collected before, including past history, present symptoms, inspection, palpation, percussion and auscultation.

Chapter IX

BRONCHO-VESICULAR AND BRONCHIAL BREATH-SOUNDS

If either of these two types of breath-sound are found, the nature of the pathology producing it, whether tuberculous infiltration, consolidation, cavitation or fibrosis, broncho-pneumonic consolidation, lobar pneumonic consolidation, abscess, bronchiectasis, infarct, tumor, atalectasis, or compression by fluid, *will have been anticipated to a considerable extent* in the inductive accumulation of the preliminary data as described above.

Bronchial breathing is too ambiguous a part of the pathology in question, too closely identical and too closely synonymous with it, too near at home to it, to serve as the starting point for a diagnosis. Using it thus and reasoning down from it tempts us to jump to conclusions and involves the risk of missing some crucial element belonging to the earlier stages of the examination. Using bronchial breathing as a starting point inevitably tempts us to look for the things that we think should go with bronchial breathing. In the first place, these things may not be present. In the second, other things, not inevitable accompaniments of bronchial breathing as such, but essential parts of the case as a whole, may be present and

overlooked. In other words, such procedure is deductive, and deduction in physical diagnosis is bad technic and bad morals. When we get to be masters, we can do as we please, but while in the process of learning an essentially inductive discipline, we must stick to inductive rules. These same remarks apply with even more force to the subject of rales.

Chapter X

RALES

Differentiation.

The geography of the rales.

1. Rales at the base, (a) bilateral, (b) unilateral.
2. Rales at the apex.
3. Generalized rales.
4. Rales over the hilum.
5. Rales of lobar distribution.
6. Rales over intermediate and unclassifiable areas.

The actual type of rale.

A. Dry rales.
   1. Sibilant.
   2. Sonorous.
   3. Fine dry.

B. Moist rales.
   1. Fine moist.
   2. Medium moist.
   3. Large moist.
   4. Consonating.
   5. Moist musical.
   6. Mucous clicks.

Respiratory phase of rales.

C. Pleural friction.

D. Muscle sounds.

"Facilis descensus Averno,
Sed revocare gradus superasque evadere ad auras,
Hic opus, hic labor est."

Virgil was doubtless thinking of rales when he wrote these lines. Rales are an easy subject to start discussing, but a mighty hard one to get out of with a whole skin. One fundamental fact to be remembered in the first place,

in the middle and all the rest of the time, is that a given rale does not connote automatically a particular type of pathology. It is the constant and strained effort to endow a particular rale with a specific and invariable significance that has caused the confusion in which the entire subject is involved. To do so is to lay down an attractive major premise from which we can reason deductively, and therefore easily. It is perfectly legitimate, if the major premise is 100 per cent. correct. But this major premise is sufficiently shy of being 100 per cent. perfect to get us into serious difficulties when we have once committed ourselves to it. It is a " facilis descensus Averno ", and more.

The most general statement that we can make about rales, that at the same time is accurate, is that rales represent either inflammation or transudation. This is not saying much, because inflammation and transudation are about all that can happen to a lung. To determine which process is present, and if the former, what kind of inflammation, we adopt three criteria, which are hereby laid down in the descending order of their importance:

I. All the data that have gone before under symptoms, inspection, palpation, percussion and changes in breath-sounds.

II. The geography of the rales, whether at the top, hilum, base, or some intermediate and unclassifiable area of the lung; whether unilateral or bilateral; whether localized or generalized; whether few or many.

III. The actual type of rale itself, whether dry, moist (fine, medium or large), sibilant or sonorous.

A fourth and often essential criterion of the significance of given rales is the x-ray, but this does not concern us here. The x-ray itself likewise must be interpreted in terms of all the preliminary data. It is becoming obvious that a true critique of physical signs bears a certain analogy to the critique which modern mathematics imposes upon Euclidean geometry. Euclidean geometry is 100 per cent. correct providing its axioms are 100 per cent. correct. Modern analysis has shown that these axioms are not above suspicion.

II. The Geography of the Rales

1. Rales at the base,
   A. Bilateral,
   B. Unilateral.
2. Rales at the apex.
3. Generalized rales (synthesis of 1 and 2).
4. Rales over the hilum.
5. Rales of lobar distribution.
6. Rales over intermediate and unclassifiable areas.

1. *Rales at the base.* — The limitation of rales to the base is first of all strong presumptive evidence against the pathology in question being tuberculous. Basal tuberculosis does indeed exist, but it is rare, and in general it cannot be diagnosed without a positive sputum.

A. *Bilateral basal rales.* — If, at the extreme lowermost

margins of the lungs, deep breathing brings out a few, very fine, dry rales, which disappear after a few deep breaths, we are dealing with the ordinary non-pathological marginal atalectasis of the over-fed and under-exercised. The incomplete ventilation of the lung-margins causes the walls of a few of the most distal vesicles to cohere. The unwonted deep inspiration pulls these opposed walls apart with resulting rales.

If, however, these extreme basal rales are more numerous and larger (fine, moist or medium moist), or more extensive than ordinary atalectatic rales should be, and if they do not clear up after a few deep breaths, they must be due to something of a more permanent nature than vesicle collapse from disuse. Such rales must be suspected strongly of being the pulmonary œdema of chronic passive congestion, the first sign of mild chronic cardiac failure, particularly in cases of hyperpiesia past middle life.

Sibilant rales or sonorous rales, at both bases, with the history of a present or recent acute cold, mean acute bronchitis. Such rales frequently are transient and may clear up on a few deep breaths or coughs. Hence you may catch them only on one side, or, if you have auscultated the chest from above downward in the usual way, may miss them altogether. For this reason it is best, in the presence of an acute respiratory infection, to listen at the bases first. Here again we see the necessity of constantly synthesizing what you are looking for now with what you have found before.

If, after an acute upper respiratory infection, basal, sibilant and sonorous, or fine to medium moist rales persist over a long period and show no sign of clearing up even after weeks, the diagnosis of chronic bronchitis generally is made, and this diagnosis is more and more coming to be considered a solecism of the worst and most pernicious kind. " C'est plus qu'une crime, c'est une faute ". The authenticity of chronic bronchitis as such is more and more being called into serious question, and the term is being relegated progressively to that group of dubious respectability of which rheumatism and malaria are classical examples.

To what then are such persistent basal rales due? First of all the situation requires a thorough nose and throat examination, calling to our aid an expert rhinologist plus the x-ray, for time and again the pulmonary pathology producing these rales is due to chronic infection of the tonsils or sinuses, particularly the latter. It is impossible to be too emphatic in urging such procedure, for no condition is more productive of ill health or more gratifyingly amenable to intelligent and radical treatment. To prescribe the usual barrels of cough-mixture, to say nothing of codein or heroin, in a condition which may be the forerunner of bronchiectasis, that most miserable of all diseases, is unspeakable. If, however, with persistent basal rales, we can find no evidence of chronic upper respiratory infection, we must think, especially in a patient of middle or later life, of the chronic passive pulmonary congestion of cardiac incompetence. But, do not say chronic bron-

chitis. These same rales, furthermore, may be all that we can find in early bronchiectasis. It may be possible to make the diagnosis only by lipiodol injection and the x-ray. It is only in the more advanced stages of this disease, than which nothing is more provocative of wretchedness, that we find the text-book *richesse* of signs and symptoms (profuse, layered, foul expectoration, clubbed fingers, large moist rales, bronchial or cavernous breathing, recurrent broncho-pneumonia and chronic invalidism). All the more reason for discovering promptly and clearing up radically all chronic upper respiratory infection in your so-called chronic bronchitis.

The sibilant and sonorous rales of asthma are readily given their correct definition by the characteristic paroxysmal attacks, the expiratory dyspnœa, the eosinophilia, the Charcot-Leyden crystals in the sputum, the history of eczema, hay-fever, urticaria and the erythema multiforme group of eruptions, and the prompt response to adrenalin. Here, again, the search for upper respiratory infection is in order, although its removal will fail frequently to cure.

If, in the presence of cough with blood-streaked sputum and fever of subacute onset, we find numerous bilateral basal fine to medium moist rales, the diagnosis is probably broncho-pneumonia. Small areas of dullness may be difficult or impossible to find, but you may be able to pick up a small area of bronchial breathing or whisper, more likely the latter, as the unobtrusive bronchial breathing

of a small patch of broncho-pneumonia might well be drowned out by the rales, the increased whisper, however, surviving. *But,* be sure that mitral or other cardiac disease is not present. Remember that what is merely a cold in the head in a person with a normal heart, may be a bronchitis or a broncho-pneumonia in a person with mitral stenosis.

If, in the presence of collapse, coma or semi-coma, and urgent dyspnœa with audible gurgling (the so-called death-rattle), we find numbers of bilateral, basal, large, moist rales at times extending well up toward the top of the chest, we are dealing with pulmonary œdema, and it is a picture of sinister import. Gradual in onset and development, it represents the terminal stage of chronic cardiac incompetence, cerebral apoplexy, nephritis, or some overwhelming febrile toxemia such as pneumonia, typhoid, tuberculosis, sun-stroke, etc. Sudden and catastrophic in its onset and terrifying in its dramatic and thunder-bolt denouement, death may ensue before diagnosis can be made. Such situations may be due to pulmonary embolism, especially in mitral stenosis, coronary thrombosis, and the paroxysmal dyspnœa of hyperpiesia. Rarely it has been described as a sudden complication of thoracentesis. Give an enormous dose of atropine and pray to the gods.

B. *Unilateral basal rales.* — Such rales must be interpreted in the light of all the other accumulated data, including the symptomatology. They may be the only physical sign of a lobar pneumonia. They are almost

certainly not tuberculous, for this disease almost invariably is located at the top of the chest. Their differentiation follows the scheme laid down under bilateral basal rales. Pulmonary œdema, essentially a basal lesion, may be unilateral in its early stages. If unchecked in its progress, however, it quickly becomes bilateral. Certain conditions, however, are essentially unilateral, although they are not essentially basal. I refer particularly to pulmonary abscess and pulmonary infarct. The former, though not necessarily basal, is most apt to be so. It is diagnosed by its concomitant phenomena, history of operation a week or so prior to the onset of symptoms, septic temperature, foul layered sputum, usually marked clubbing of the fingers, possibly dullness, possibly a few moist rales and generally diminished breath-sounds. If the abscess has become chronic and is draining well, there may be no fever. In a case demonstrated to the students, the only chest-signs were marked dullness at the left base, diminished tactile fremitus and almost absent breath-sounds with a few very fine moist rales near the upper margin of dullness. The students made a diagnosis of pleural effusion. So far as chest-signs went, this diagnosis was perfectly consistent with the findings mentioned, but the students did not take into account the fact that the mediastinum was displaced toward the lesion instead of away from it, nor did they notice the clubbing of the fingers.

Pulmonary infarct is essentially a unilateral lesion, unless, of course, the emboli are multiple. It is not essen-

tially a basal lesion, but may be so. Its diagnosis rests on a possible source of emboli (particularly mitral stenosis), a sudden onset, almost always with pain, not necessarily catastrophic or rapidly fatal but sometimes so dependent upon the extent of embolism and resultant shock, fever for a few days, sometimes high and septic, a few localized rales and possibly a pleural friction. Sterile emboli, if not promptly fatal from shock, end in recovery in a short time. Septic emboli terminate in abscess. It is appropriate to inquire into the source of the pulmonary emboli so frequent in mitral stenosis. To this question the student invariably answers, and very promptly, "from the mitral vegetations". At the next question, "How do they get to the lung?", there is a painful and embarrassed silence. We will leave it to the ingenuity of the student to figure out whence cometh the pulmonary emboli of mitral stenosis.

2. *Rales at the apex.* — Transient sibilant rales at the apex, which clear up after cough, mean nothing but bronchitis of the medium-sized tubes. The fact that such rales may not be heard over the rest of the chest, ordinary bronchitis possessing such general distribution, usually means that they have been present, but have cleared up while you were listening to the apex. If, however, apical sibilant rales persist, and no similar rales are heard over the rest of the chest, then think of the possibility of localized bronchitis, never forgetting that localized bronchitis is apt to be tuberculous. However, such persistent, localized, apical, sibilant rales are merely suspi-

cious, not diagnostic, of tuberculosis. In their presence other data are needed for a diagnosis.

Fine dry, or any sized, moist rales, at the apex, if persistent after cough, or if brought out by cough, mean tuberculosis. If such rales are moist, there may be demonstrable some change in percussion-note or breath-sounds. Here, however, the inevitable "exceptis excipiendis" comes in. Very rarely such rales may, like localized harsh breathing, follow the grippe and last for several weeks, causing disastrous mistakes in diagnosis. Without a history of grippe, they are certainly tuberculous. And even with a history of recent grippe, tread softly, for the grippe probably was not grippe, but rather tuberculosis masquerading in sheep's clothing. In the overwhelming majority of cases such rales mean tuberculosis. If tuberculosis is suspected, they must be searched for with the greatest care.

3. *Generalized rales; (a) sibilant; (b) moist.* — Generalized sibilant rales mean bronchitis or asthma. Do not forget, however, that underneath the sibilant rales, and masked by them, there may be other pathology of a more serious nature, in particular, tuberculosis. The only way to protect yourself from the grievous error of missing the latter, is to train yourself into an attitude of chronic suspicion regarding the disease. The doctrine of the parsimony of causes, i.e., the rule of scholastic logic which teaches us to look for single rather than multiple explanations of a phenomenon, may be all right in philosophy, but it is dangerous in diagnosis. Always watch out for

alternative possibilities. Generalized sibilant rales merely mean bronchitis. They do not rule out tuberculosis merely because they mask it. Such rales, if numerous and obtrusive, very often absolutely preclude any chance of diagnosing tuberculosis, either positively or negatively. If the history has shown anything of ominous suggestiveness, take an x-ray, and, of course, always, several sputum examinations. Physical examination, *per se,* is, in this situation, often hopelessly baffled.

These remarks apply, with even more truth, when we are dealing with generalized moist rales. Everything, even changes in breath-sounds, may be drowned out by the overwhelming tumult of the rales. The history, percussion and the whispered voice may help, but time and again, with this plethora of rales, we must fall back on the x-ray. Sputum examination is so obviously a matter of course, that no more than mere mention of it is needed. Some have said that the hardest types of tuberculosis to diagnose are the very early and the very advanced cases.

To sum up, generalized rales are both of the apex and of the base. Their differentiation must follow the criteria laid down for both of these localities.

4. *Rales over the hilum.* — Sibilant rales in this locality mean bronchitis. Fine dry or moist rales, if they are limited to the hilum and do not extend into the lower lobe, are almost certainly tuberculous. If they extend into the lower lobe, *without extending toward the apex,* they are less likely to be tuberculous. In this event, look for chronic upper respiratory infection, particularly of the

sinuses. If they extend up toward they apex, they are even more likely to be tuberculous than if limited to the hilum alone. Remember that in a small area limited to the hilum, you will probably get no help from the x-ray, for everything will be overlaid by the dense shadow of the mediastinum. Diagnosis rests on general clinical considerations, including history and *repeated* sputum examination, plus the all-important fact that rales limited to the hilum are most probably tuberculous. Negative sputum examination is of no value whatsoever.

5. *Rales of lobar distribution.* — These must be differentiated according to the criteria laid down for apical and basal rales.

6. *Rales over intermediate and unclassifiable areas.* — Not even an approximate rule can be laid down. They must be diagnosed in terms of every bit of available data.

III. The Actual Type of Rale

The descriptive classification of rales is a difficult and dubious subject. The difficulty lies not in telling what is so, but in avoiding saying what is not so. There are two definite psychological hazards which tend to confuse the issues right at the start. First, the inherent difficulty of giving an unambiguous description to sounds upon which several observers may differ legitimately. Second, the ever-present temptation, for the sake of saving mental work, to try to correlate a given type of rale with a definite type of pathology. From the very start the existence of

these two hazards must never be forgotten. The classification of rales and the interpretation of the rales in terms of pathology always must be to some extent provisional.

A practical tentative classification of rales and adventitious sounds can be laid down according to the following scheme:

A. Dry rales.
B. Moist rales.
C. Pleural frictions.
D. Muscle sounds.

A. Dry Rales

1. Sibilant rales.
2. Sonorous rales.
3. Fine dry rales.

First of all, remember that there is no such thing as an actual *dry* rale. When we speak of dry rales, we mean rales that "sound" dry, in contradistinction to moist rales, which "sound" moist.

Sibilant and sonorous rales. — Sibilant rales are musical chirps and squeaks of a dry non-sticky quality. Sonorous rales are non-musical snores and grunts. Sibilant and sonorous rales result from swelling of the mucosa of the large and medium bronchi with resulting narrowing of the lumen. They are, therefore, signs of bronchitis of the large and medium tubes, the ordinary, common or garden variety of bronchitis. Bronchitis, however, may occur without their being present. They also occur in asthma. They are, therefore, associated with lesions which, *per se*, do not cause changes in percussion-note

or breath-sounds. But remember that lesions causing such changes may be present independently and be completely masked by the noisy sibilant and sonorous rales. As bronchitis usually is a bilateral disease, these rales are heard bilaterally, and as general bronchial congestion usually is more marked at the base, they tend to be more in evidence in that location. But, as they tend to disappear after cough and deep breathing, they may have cleared up temporarily during your auscultation of the apices, and you may, therefore, miss them when you reach the base. For this reason, when you suspect ordinary bronchitis, it is best to auscultate the base first.

Fine dry rales. — These can be imitated fairly well by rubbing a few hairs between the thumb and forefinger. If few in number and well separate from each other, they are referred to by some writers as "dry crackles", or "isolated crepitant rales". If many in number and occurring in small showers, they are known in the textbooks as "crepitant rales". The difference between dry crackles and crepitant rales is simply numerical. They are almost invariably limited to the inspiratory phase, very rarely appearing in the expiratory, and frequently require for their demonstration the cough at the end of expiration, a manœuver which always must be carried out. Now then, what do they mean? First of all, they are heard frequently at the extreme base posteriorly, or at the lower margins of any of the lobes, during the first few deep inspirations, after which they generally disappear. They are, in this situation, very certainly due to the

separation, by deep inspiration, of the opposed coherent surfaces of a few unused air vesicles on the extreme confines of the lung. Ventilation of these vesicles causes first the appearance and then the clearing up of these rales. In this situation they are referred to often as atalectatic or marginal rales. They are, however, true fine dry rales, and in other situations of the chest will have an entirely different significance. In the presence of pathology, they probably represent early pneumonic infiltration, the term "pneumonic" being used in its generic sense. At the apex they are presumptive evidence of tuberculosis. In other parts of the chest, *with appropriate symptoms,* they may be the earliest signs of lobar pneumonia. The absolute acoustic identity of fine dry (crepitant) and atalectatic marginal rales has tempted some speculative souls to explain their incidence in early tuberculosis as follows. A very early tuberculous bronchiolitis, which as yet has not reached the stage of free exudation, causes an occlusion of the bronchiole in question through swelling of its walls. The lack of ventilation of the vesicles distal to this bronchiole causes them to lose their contained air through absorption. Their walls collapse and cohere, become atalectatic in other words. The cough forces air through the occluded bronchiole, reinflates the vesicles tributary thereto, and separates their walls, with the production of fine dry rales identical in character and mechanism of production with the marginal rales so frequently heard at the base of the normal lung. The fine dry rales of early tuberculosis, in short, are produced not at the site of the

actual pathology, but distal to it. Now this is all very fine, but it is probably not true. This very plausible and attractive theory is very interesting to the metaphysical side of our nature, but it completely disregards the fundamental difference between marginal, non-pathological, fine dry rales and apical, tuberculous, fine dry rales, which is that the former are transient, the latter persistent. Later, when we come to take up the signs of tuberculosis, we will see that the fine dry rales of early tuberculosis are probably an outgrowth of a still earlier sign, granular breathing, this last being a sign which conceivably can only arise in the actual site of pathology. In other words, the fine dry rales of early tuberculosis are not indirect, but direct evidence of disease, and though commonly referred to as an early sign, actually mean that the stage of open exudation has been reached.

B. Moist Rales

By moist rales we mean rales that "sound" moist. The distinction between dry and moist rales is much easier to make out with the stethoscope than it is to describe in words. Moist rales are generally described by the student as having a sticky, wet, or "gooey" sound, and this is precisely what is meant.

We divide moist rales into:

1. Fine moist rales.
2. Medium moist rales.
3. Large moist rales.
4. Mucous clicks.

1. *Fine moist rales are* slightly larger than fine dry rales. They can be imitated fairly well by opposing and separating the balls of the thumb and forefinger after moistening them with molasses. They are usually referred to as subcrepitant, a term which should be relegated to limbo.

In general it may be said that moist rales represent a more advanced grade of exudation or transudation than do dry rales.

2. *Medium moist rales* are larger than fine moist rales. In general, the larger the rale the more pronounced the process, whether exudation, transudation, or caseation. Between these three processes, however, there is obviously a vast diagnostic difference, and since a medium moist rale may connote any one of the three, it is obvious that, except in very general terms, we cannot rely for our diagnosis on the mere definitive characteristics of the rale itself, but must refer to all the other data that have already been gathered, to wit, history, palpation, percussion and breath-sounds. The average medical student seems to feel that he has not gotten his money's worth out of a clinical session unless he has heard some rales, and when he does hear them he shows an expansively naïve delight at having, at last, found one clear-cut thing in a desert of obscurity. Remember that rales, although of the greatest importance, must, for the sake of developing technic, be relegated to a secondary position and be regarded for the most part as merely complementary to the other data.

There are two special types of moist rales which must be mentioned here. At times fine to medium moist rales

will have a high pitch and a curious, incisive, clear-cut, snapping quality, something like that of a toned-down telegraph receiver. These are known as consonating rales, and they derive their special characteristics by participating in the acoustic qualities of the tissue surrounding the site of their origin. You will remember that bronchial breathing is high-pitched and clear-cut. The similar characteristics of the consonating rales spring from the fact that these rales arise within tissue potentially capable of producing bronchial breathing, to wit, consolidation or cavitation, especially in broncho-pneumonia and caseating tuberculosis. Their presence signifies a high degree of intra-bronchial exudation, surrounded by consolidation or cavitation. Actual bronchial breathing, to be sure, need not be present, the bronchophonic characteristics being manifested solely in the rales. Given such rales, with no perceptible changes in breath-sounds, the presence of pathology corresponding to bronchial breathing can be inferred with considerable accuracy.

The second type of moist rale I have never seen described in any text. It has all the characteristics of the consonating rale, except that it is always medium in size, never fine. Its unique feature is its musical, chirping quality, precisely like that of the ordinary sibilant rale. It is distinguished from the latter, however, and this is most important, by being moist in sound. It occurs not infrequently over caseating tuberculosis. I have never heard it anywhere else. It must not be mistaken for the sign of a banal bronchitis.

In is unwise to put too much weight upon the phase in which the rales appear, whether inspiratory or expiratory. It is true that fine dry rales and the earliest fine moist rales are almost without exception limited to inspiration, and that, in general, an extension of the rales into the expiratory phase connotes a greater advancement of pathology. However, a pleural friction, which often indicates a comparatively insignificant or even healed process, quite frequently is audible in both inspiration and expiration, and, which brings us to the point in question, quite often is indistinguishable even to the ear of the expert from fine dry, or even fine moist, rales. In such a situation, if we put too much emphasis on the phase of incidence of the rales, the presence of adventitious sounds in the expiratory phase might lead us to the serious error of diagnosing a graver situation than actually existed. Take, for example, an extensive patch of inspiratory and expiratory fine dry and fine moist rales at the lateral or posterior base. How are we to distinguish between a comparatively trivial dry pleurisy, perhaps a healed dry pleurisy, from a far more significant intra-pulmonary lesion? By the history, by the symptoms, by the presence or absence of clubbing of the fingers, by the patient's general health, and, alas! by the x-ray. The greater the discrepancy between the negative symptomatology on the one hand and the prominence of the rales on the other, the more apt is the lesion to be extra- rather than intra-pulmonary.

3. *Large moist rales* mean the presence of a high degree of very liquid exudation or transudation, such as might

be expected in resolving pneumonia, advanced caseation or pulmonary œdema. Their significance is determined by the nature of the other data.

In searching for fine dry or fine moist rales, never fail to have the patient give a slight cough *at the end of expiration,* and after coughing, breathe in. This manœuver frequently will bring out rales otherwise hidden. They will make their appearance immediately after the cough or at the end of the next succeeding inspiration. The medium and large moist rales generally do not need the cough to bring them out. The cough frequently will demonstrate a valuable sign of cavity known as the post-tussic suction. Have the patient cough at the end of expiration, but this time have him hold his breath after the cough. Immediately following the cough, *and while the patient is holding his breath,* there will be heard what sounds like a short, high-pitched, blowing breath-sound, which, occurring while the patient is not breathing, must be due to some other mechanism. The cough compresses the walls of the cavity. Immediately after the cough, these walls, if sufficiently elastic, rebound to their original position, sucking air in after them, which sucked-in air gives rise to the sound in question.

If the rales are so noisy as to obliterate the breath-sounds, as frequently happens, try the whispered voice. This may bring to light an unsuspected situation the equivalent of broncho-vesicular or bronchial breathing, such as infiltration, consolidation, cavitation or fibrosis.

4. *Mucous clicks.* — The mucous click is a rather large

and very sticky moist rale. Its size and wetness have a rather ominous sound and suggest advanced caseation. It is due, however, to mucus in the small bronchioles around a healing tuberculous focus, and in fact may persist long after the actual tuberculosis is healed. It is, in short, a not unfavorable sign. It should always be checked by the x-ray to rule out an undemonstrable parenchymatous involvement. When the examiner hears it, he generally shakes his head in a learned sort of way and announces profoundly and gloomily, " There is moisture present ", which is sufficiently non-committal. As a matter of fact the fine dry rale is infinitely more sinister, because it represents the inception, not the end, of a tuberculous process.

C. Pleural Friction

Most of the ammunition for this subject was expended in the previous paragraph devoted to the significance of the phase during which rales appear, whether inspiratory or expiratory. The main diagnostic implications are discussed there. The absolutely typical pleural friction is a dry, leathery, shuffling, back-and-forth sort of sound, possessing a much higher degree of continuity than rales, not influenced either positively or negatively by cough and heard in both respiratory phases. Unfortunately there are many exceptions to this cut-and-dried text-book description, and these exceptions are discussed above. At times the sound cannot be distinguished from fine, or even medium, moist rales. The mid-axillary region is the

usual site in which to hear friction-rubs, as here is the greatest amount of free play between parietal and visceral pleura. But they may occur anywhere, not infrequently indeed at the apex. Hearing them in this locality, it is a nice question to decide whether you are dealing with the extra-pulmonary signs of an old and obsolete tuberculosis, or the intra-pulmonary signs of an early and advancing tuberculosis.

D. Muscle Sounds

There is one perennial source of confusion, namely, muscle sounds. The student need not worry, however, because at times even an experienced man will mistake them for rales. Muscle sounds are a deep vibrant rumble, with a marked continuity, different from the sharply discontinuous, staccato explosions of rales, and they last all through the inspiratory and expiratory phases. In fact they seem to last beyond the expiratory phase, through the interval when the patient actually is not breathing at all, over into the next inspiration. They are noisy and exasperating, but it is possible to cultivate a "deaf-spot" for them. They are less likely to occur in patients with a well-developed panniculus adiposus, which seems to blanket them off, and hence are less bothersome in women. Joint-crepitus often causes sounds very similar to rales and must be guarded against. A useful manœuver is to have the patient breathe in short gasps or sighs. This frequently will render less obtrusive both joint-crepitus and muscle sounds.

Chapter XI

THE SIGNS OF TUBERCULOSIS

Sputum examination.
Pleurisy with effusion.
Significance of hemoptysis.
Apical moist rales. Infrequent sequel of influenza.
X-ray findings.
Other symptoms.
Percussion.
Auscultation.

"A wicked and adulterous generation seeketh after a sign, and there shall no sign be given unto it."

This, the most profound of all the verses in the Bible, is directed against those of the human race, the vast majority, incidentally, who think that merit is to be acquired by some cheap and easy formula, rather than by prayer and fasting. It is highly applicable to the diagnosis of tuberculosis. There is no cheap and easy route. A positive sputum is, of course, a sure sign, in the Biblical sense, but it means a pretty well advanced process. Finding a positive sputum is not diagnosing tuberculosis. Any laboratory technician can do that. Diagnosing tuberculosis means the discovery of the process in the minimum of time so that the patient can have the maximum of chance. Tuberculosis is diagnosed by a skilled technic plus a peculiar synthetic discipline of the mind. This technic and this synthetic inductive discipline I have

attempted to describe in the preceding pages. Dealing with the detached topic of tuberculosis, it is necessary for descriptive purposes to abandon the inductive to a certain extent and resort to a more deductive method of approach. In the present chapter I will not differentiate between signs and symptoms, but will attempt to treat the subject as an organic unit.

The first part of the equipment necessary for the early diagnosis of tuberculosis is an unremitting, unrelenting, inexorable attitude of strong suspicion. Always think of tuberculosis. A young girl came to New York to buy a wedding dress. She consulted me about a cough, which she had had for a year. Her local doctor had squirted out her throat twice a week for twelve months. He had never looked at her chest. Examination showed innumerable fine and medium moist rales over both upper lobes, about twenty bacilli to each microscopic field and in the x-ray extensive parenchymatous consolidation and cavitation. The wedding invitations were called in. Now it may not be considered good literary form to indulge in what the art critics call "morbidezza" in a technical medical discourse, but this sort of thing is happening all the time, and all for the same reason, lack of suspicion. There actually seems to be what the psychoanalysts call a "resistance" against the idea of tuberculosis. It is almost as if "nice people" did not get tuberculosis. Well, nice people do get tuberculosis, and nice people get syphilis too, another disease which is missed as a result of insufficient evil-mindedness on the doctor's part. Always suspect,

but do not make a definite diagnosis on suspicion alone. Such a diagnosis, fraught as it is with the most serious consequences to the patient, must be based on a convincing array of evidence. Voice your suspicions, if you must, without unduly disturbing the patient, but do not subject him to a revolutionary change in his life until you are sure.

Sputum Examination

A positive sputum is an inevitable sign of tuberculosis. But be sure that the positive sputum is really positive. One single lonesome bacillus in fifteen minutes' search is pretty thin evidence unless the finding is duplicated in later specimens. It may be an artefact. Now, when should the sputum be examined for tubercle bacilli? The answer is, whenever there are cough and expectoration. It is very disconcerting to have an apparently ordinary lobar or broncho-pneumonia turn out to be tuberculous, and if to such a case you should have given pneumococcus serum or antibody, thereby stirring up the animals to the point of disaster, it requires a high degree of rhetorical skill to rationalize your way out of it. If, in such a situation, the patient's family say, "Doctor, did you examine the sputum?" and when you answer, "No", they continue with the question, "Well, why not?" or words to that effect, it is hard to know what to say. This procedure, so simple, so direct, so specific and confoundedly obvious, is postponed time and again and always for the same reason, a low index of suspicion *in re* tuberculosis.

A husky young city fireman came into the Bellevue Tuberculosis Clinic. His complaints were rather marked dyspnœa on exertion, very slight cough, very slight expectoration and a pulse of 100 with a normal temperature. Physical examination and the x-ray disclosed absolutely nothing, and I was stumped for a diagnosis, being perhaps inclined toward the dubious but convenient diagnosis of myocarditis, convenient, because no one can disprove it. I was considerably chagrined when the sputum was reported loaded with tubercle bacilli. Now here was where the wicked generation seeking after a sign scored on the toilers in the vineyard, and I breathed a prayer of gratitude that in the Bellevue Tuberculosis Clinic the sputum is examined as a routine, for I do not think it would have occurred to me to have done it myself. The answer is simple. My index of suspicion was not up to scratch.

Now what does a negative sputum mean? A single negative sputum means absolutely nothing, no matter what the attending circumstances. Repeated negative sputa, say a dozen or so, in the presence of very slight pathology, likewise mean absolutely nothing. However, repeated negative sputa, *in the presence of considerable pathology, and with considerable expectoration,* are strong evidence against tuberculosis, but only with these attending qualifications. The whole situation reminds one of Nigger Jim's conversation with Huckleberry Finn on the signs of bad luck, of which, you will remember, he recounted quite a catalogue. When Huckleberry asked, "Well, what are the signs of good luck?" Nigger Jim

replied, "Huck, I jex' reckon they ain't no signs of good luck." This is the attitude to take toward all negative laboratory findings, and the earlier this attitude is acquired, the better.

Pleurisy with Effusion

Pleurisy with effusion means pulmonary tuberculosis no matter if specific physical signs and x-ray findings are negative. Pleurisy with effusion is a specific physical sign of tuberculosis, and woe unto the physician who fails to accept it as such. Woe, also, unto his patient! The books say that 80 per cent. of all cases of serous effusion are tuberculous. But don't pay any attention to such a figure. Serous effusion actually means tuberculosis in about 100 per cent. of cases. It is a real, sure-enough, honest-to-goodness "sign" and should be sufficiently convincing to any generation, no matter how wicked, etc. Of course, serous effusion must be distinguished from other forms of pleural fluid, such as the non-inflammatory transudates of cardiac and renal disease, the sero-fibrinous pleurisy of pneumonia and empyema. These last will be distinguished by their physical and microscopic characteristics and also by their typical, concomitant symptomatology. Sterile empyema is almost invariably tuberculous.

Significance of Hemoptysis

Hemoptysis *almost* inevitably means tuberculosis. Ignoring this fact has occasioned innumerable tragedies. But, an hemoptysis must be an hemoptysis. A speck or minute streak of blood in scant sputum after violent coughing is not an hemoptysis. In such cases the throat-mirror will often reveal a fat cauliflower-like lingual tonsil with a lot of varicose veins. With luck, a small hemorrhage from one of these veins may be actually seen, if sufficiently recent. But a half a dram of free blood, particularly if full of air-bubbles, is a genuine hemoptysis. Having thus established the fact of hemoptysis, we automatically infer tuberculosis, but we are not yet in position to fill out an application card for a sanitorium. For the mere hemoptysis tells us nothing about the actual status of this particular case of tuberculosis, whether in the past, present, or future tense, whether early or late, whether obsolete or advancing. Consider the pathology of hemoptysis. It may be the initial symptom of an early active process in a supposedly healthy person, in which case it arises from a small area of ulceration in a minimal parenchymatous process. In such a situation it means *Danger,* and if this unequivocal signal be heeded, fortunate is the patient, for he will get on the job at once, mobilize his forces and get cured. If neglected, — !

An hemoptysis occurring in a case already demonstrated to be tuberculous may signify one of three distinct things. It may be the equivalent of the above situation and

represent the early evidence of an advance of the old process into new tissue. It may come from the erosion of an artery in a cavity, in which case the hemorrhage may be acute and menacing. This is the frequently fatal type. Lastly it may arise from the sloughing out of a calcified area in a completely healed, inactive and obsolete lesion, and thus carry with it no menace at all. But one must tread softly in making such a diagnosis. Such a conclusion must be made with considerable caution and with the use of the best clinical judgment. In other words, although practically every hemoptysis (for there are exceptions) means tuberculosis, yet every hemoptysis has a meaning all its own and must be interpreted inductively in the light of all the data, history, physical examination, pulse-temperature record, weight and strength record and x-ray.

Apical Rales

Persistent fine moist rales at an apex are practically pathognomonic of tuberculosis. Yet even this so sure sign must be checked by other evidence, so serious is the question at issue. Localized moist rales at an apex mean localized apical bronchitis, and localized apical bronchitis means tuberculosis in the vast majority of cases. Yet even to this rule there may be the rare exception. In the influenza epidemic of 1917–1918 and 1918–1919, there were occasional cases in which localized apical bronchitis occurred, manifested by a small patch of fine moist rales. The natural and inevitable diagnosis was tuberculosis,

but the point is that the rales cleared up in a few days, and careful follow-up with the x-ray and repeated physical examination disclosed nothing.

X-ray Findings

Lastly come the x-ray findings, but this is a subject apart, highly complex and full of variables. Suffice it to say that the only pathognomonic x-ray sign of early tuberculosis is the characteristic shadow of parenchymatous infiltration. To diagnose tuberculosis on the basis of so-called peribronchial infiltration is skating on thin ice. And yet, on these very meagre grounds, enthusiastic radiographers are diagnosing tuberculosis with a temerity and enthusiasm that would be admirable, were the diagnoses only correct. It is almost as if they were trying to show how good they were, how superior to the poor devil of a clinician with his crude percussion and still cruder auscultation. For, to diagnose tuberculosis on the basis of peribronchial infiltration is wrong. Peribronchial infiltration may be based on purely non-specific causes, such as chronic tonsilitis or chronic sinusitis. The usual conventional hedge is, "Yes, peribronchial infiltration is tuberculosis, but it is not *clinical* tuberculosis." But, in the name of Heaven, about what is it that we are vexing ourselves, if not clinical tuberculosis? Everyone has "ein bischen" of "unclinical" tuberculosis, to coin a new word, and peribronchial infiltration does not even

mean "unclinical" tuberculosis. Check your x-ray findings with your history and physical examination.

Other Symptoms

Such are the pathognomonic signs of tuberculosis, positive sputum, serous effusion, hemoptysis, persistent apical fine dry or fine moist rales and the x-ray shadow of parenchymatous infiltration, I trust duly emphasized, and, I trust, duly qualified. Outside of these pathognomonic signs, the diagnosis of tuberculosis is purely inferential, and on the degree of probability depends the course to be followed, whether watchful waiting or complete limitation of the patient's activity. So serious is the latter step, however, not only because of its depriving the patient of earning capacity, but also because of its sinister effect on his morale, that the fixed policy should be that of an optimist, not, however, of the optimist who has been defined as a person who does not know what is coming to him, but the optimist who has the courage derived from knowledge. Chronic cough, fatigue, loss of weight and amenorrhœa, without obvious cause, require explanation, but too much must not be inferred from them. Chronic cough, without chest signs, may be based on a number of things, particularly upper respiratory infection, such as chronic tonsilitis or chronic sinusitis. An enlarged lingual tonsil is a very frequent cause of chronic cough, and one which is almost as frequently overlooked. This cough usually is characterized by its insistent violence, a violence which is out of all proportion to the meagre expectoration

it produces. Not infrequently it is associated with a sense of irritation referred to the substernal region, thus giving rise to the false impression that a tracheitis is present. All the cough medicines in the world will have no effect on it. Pulling the patient's tongue forward and looking down with a throat-mirror discloses the large cauliflower-like lingual tonsil, which, acting like a foreign body, causes violent attempts to get rid of it. The cure is simple and effective and has the added value of being a good therapeutic test. A few applications of concentrated ferric chloride solution will shrink up the lingual tonsil and cure the cough. Some care is needed, for if any of the solution drips onto the vocal cords, considerable unpleasantness will ensue. The patient pulls his own tongue out, leaving two hands for the doctor, one for the throat-mirror, the other for the applicator.

Fatigue and loss of weight and strength may be due to many causes, particularly focal infection, mucous colitis, syphilis, thyroid disturbances, psychoneurosis and retention of metal, especially arsenic. The last named deserves special mention as recently it has been suspected to be of wide-spread occurrence. As arsenic occurs in demonstrable quantities in an article of universal consumption, commercial glucose, its role as chronic intoxicant is sufficiently frequent. It is a sympathetic nerve poison, and hence may give rise to any of the symptoms of vagotonia, which, by the way, frequently occur in tuberculosis. Its more specific manifestations, manifestations which should always give rise to a suspicion of arsenic retention, are

eczema, blepharitis, œdema of the lids, and a curious blood-chemistry picture which has come to be regarded as probably pathognomonic, namely, a high blood-sugar and a low blood-chloride. Remember that hyper thyroidism may be associated with loss of weight and rapid pulse. If these symptoms are associated with an afternoon rise of temperature or pulse-rate, they take on an added and sinister significance, and the general situation may force a tentative diagnosis of tuberculosis even without the pathognomonic signs. You are between the Scylla of early diagnosis and the Charybdis of correct diagnosis and may the gods be with you.

We will now attempt to follow the changes in physical signs corresponding to the changes in the underlying pathology. It is not possible to establish a complete correlation between them. Attempts to do so always lead to a certain degree of arbitrary rationalization and this factor of error must be kept in mind always. The natural desire to explain things, to establish relations, tempts one to plausible theories which may not be strictly true, and the natural desire to link up signs and pathology is fraught with this risk. In the following description, bear in mind that the relationships, however arbitrarily expressed, carry with them a tacit qualification.

I. Percussion

The earliest lesion consists of a few small scattered tubercles at the apex. Corresponding to this there is

usually not true dullness, but a slight decrease in the duration of the note with a slight elevation of pitch. As the tissue surrounding the tubercles may be relaxed, there may be a corresponding slightly tympanitic or hyperresonant overtone, which may, but usually does not, cover up the slight impairment of resonance. If the slight tympany predominates over the slight impairment we may be misled into placing the presumptive lesion on the wrong side. These slight percussion changes are the very earliest signs and precede any auscultatory changes. They require for their recognition the most careful technic, even to the percussion of the two apices during the same phase of respiration. They are too fine-spun to have any pathognomonic value, but in the presence of suspicious symptoms pick up an added diagnostic momentum.

A slight retraction of the inner border of the apex can, in this stage, be demonstrated with more assurance. It is not due to fibrosis, which has not yet appeared, but to lessened functional activity, and with early cure may disappear. Impaired resonance over one aspect of an apex, anterior or posterior, may be accompanied by hyperresonance over the other aspect. So, finding one, look for the other.

Actual dullness is not an early sign because it means that a fairly good-sized area of consolidation has developed. In percussion the apex must be divided into three zones, each of which is to be compared separately with the corresponding zone on the other side. The innermost

zone shows a slight degree of tympany, derived from the closely adjacent trachea. The outermost zone shows slightly impaired resonance as a result of the underlying muscles. The central zone gives the lung tone, normally slightly modified of course on the right in the direction of impairment of resonance and heightened pitch.

The commonest sites of dullness are the inner part of the supra-clavicular fossa, the inner third of the infra-clavicular space, and the supra-spinous fossa. Percussion of the clavicle itself to bring out the dullness of the lesion which at times occurs directly under the bone is unreliable.

Tympany accompanied by dullness is not necessarily indicative of cavity. As mentioned above, it may be due to relaxed tissue adjacent to a small area of infiltration. This tympany, unlike that of cavity, does not show Wintrich's sign (higher pitched percussion note with the mouth open) and may be thus distinguished. Unduly hard percussion at the apex may arouse tympany from the large bronchi and may lead to error.

With the advent of marked dullness, due to infiltration advancing toward consolidation, comes increased resistance to the finger on percussion, and the retraction of the apex border becomes more definite. Such marked dullness generally runs down from the outer end of the clavicle to the upper part of the sternum, forming a triangular area, the advancing border of which peters out into normal resonance. Below the area of dullness the lung is apt to be hyper-resonant, either from relaxation of tissue or from

hyper-function. Outlying areas of dullness frequently occur in the fourth intercostal space. On the left, such an area may be found just outside the border of the heart. With advance of the disease retraction of the base occurs, with limitation of the motion of the diaphragm on the affected side. This is easily demonstrated by marking the lower limits of resonance in three phases, quiet breathing, deep inspiration and deep expiration.

II. Auscultation

The earliest auscultatory changes are inspiratory, and to detect them, single-phase auscultation should be used, that is, the separate comparison of successive inspirations in symmetrical spots, without listening to the intervening expiration. The following are the earliest changes in the order of their appearance:

1. *Granular breathing.* — This is rough and low-pitched and is made up of a succession of very short sounds very closely spaced, without definitely distinguished intervals. It is probably due to slight narrowing or uneven surface of the bronchioles, and this always means parenchymatous trouble. It is entirely different from harsh or puerile breathing. It is most common in the following sites, in the order named: (1) the supra-spinous fossa, (2) the clavicule-sternal angle, (3) the supra-clavicular fossa, (4) the advancing border of disease. Muscle-sounds must not be mistaken for granular breathing.

2. *Diminished breathing* comes next in order, but is of less value diagnostically. It is due to obstruction by tubercles or mucus. It is of vesicular or slightly granular quality, and inspiration is feebler than expiration. To suggest tuberculosis, it must be limited to the apex and be persistent after cough.

3. *Cog-wheel breathing* generally is limited to the inspiratory phase. The respiratory murmur is divided into short periods separated by pauses, giving a sort of soft shuffling sound. Various guesses as to its cause are (1) valve-like action of swollen mucous membrane or secretion in the smaller tubes, (2) uneven contraction of the lung from the presence of tubercles, or (3) the rubbing of sub-pleural tubercles. It may be simulated by (1) muscle-sounds, (2) adhesive pleurisy, (3) pain, and (4) heart-action. Diagnostically it is in no sense final, but may carry a certain suggestiveness when combined with other evidence. To have any value at all it must be limited to a small area. It is best heard during quiet breathing. It is less common at the apex than in the clavicular-sternal angle, at the right base behind, or on the advancing border of evident lesions.

4. *Harsh breathing* often is confused with granular or rough breathing. It is dry, sharp and more blowing, and the breezy softness of the normal respiratory murmur is lacking. It is generally more marked in expiration and frequently is limited to that phase. It does not represent the earliest change in the development of the pathology, but is accompanied by dullness and represents the transi-

tion to broncho-vesicular and bronchial breathing. While not absolutely diagnostic of tuberculosis, it is, if heard on quiet breathing and limited to the apex, of considerable value. Two exceptions must be noted. It may persist for weeks after the apical pneumonia of influenza, and to a certain degree it may be normally present at the right apex. Thus it is of greatest value when heard at the left apex. The harshness is due to narrowing of the bronchi and condensation of the lung tissue. Unlike granular (rough), feeble, or cog-wheel breathing, it is heard most often at the extreme apex in front.

Prolonged expiratory sound without harshness is of little significance. Puerile breathing is not at all reliable. Undue transmission of the heart-sounds to the right apex is of considerable value, unless the heart is beating very hard.

5. *Rales* are not strictly incipient signs. They represent an advance beyond the signs considered above, and with them we reach the stage of *definite* auscultatory diagnosis. They likewise have their embryology and develop in the following succession:

(a) *Dry crackles,* or "isolated crepitant rales", are minute dry cracklings, three or four at the most, each well separated from the other and almost invariably limited to inspiration. They are permanent and are not removed by cough. At the apex they are presumptive evidence of tuberculosis.

(b) *Crepitant rales,* or fine dry rales, sound dry but are not strictly dry, as they are probably caused by the

separation of alveolar walls which are stuck together by secretion. This is certainly the mechanism of their production at the base, where they frequently occur in normal persons on deep breathing and then are referred to as atalectatic or marginal rales, and this is probably the mechanism when they occur at the apex. In the latter situation they represent small areas of pneumonic infiltration. Acoustically the same as the marginal atalectatic rales heard at the normal base, they do not clear up on deep breathing or on coughing, as do the latter, but persist. In fact, the cough often brings them out by explosively separating the coherent alveolar walls. They occur almost invariably in inspiration and are precisely the same as dry crackles, except that they are more numerous and occur in showers or salvos. At the apex they mean active tuberculosis.

(c) *Fine moist rales,* badly denominated "subcrepitant rales", are not limited to inspiration, but may invade the expiratory phase. They represent a further advance toward softening, but may occur in a healed or inactive area. The fine dry rale, however, invariably means activity.

For the diagnosis of early tuberculosis, rales must be strictly localized, usually at the apex, and persistent.

Conclusion

The goal of tuberculosis diagnosis is early diagnosis. Pathognomonic signs, even those conventionally denomi-

nated early signs, are not strictly early, for they represent a definite advance beyond the stage of actual beginning. True early diagnosis is a synthetic, not a rule-of-thumb, judgment and is based on all the available data. Quite frequently the only data available are those pertaining to the history. They must be interpreted in the light of a mature point of view, brought into high relief by an attitude of unrelenting suspicion, shaded by an attitude of critical caution.

Y0-AVZ-613